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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	SEP 01	New pricing for the Save Answers for SciFinder Wizard within STN Express with Discover!
NEWS	4	OCT 28	KOREAPAT now available on STN
NEWS	5	NOV 30	PHAR reloaded with additional data
NEWS	6	DEC 01	LISA now available on STN
NEWS	7	DEC 09	12 databases to be removed from STN on December 31, 2004
NEWS	8	DEC 15	MEDLINE update schedule for December 2004
NEWS	9	DEC 17	ELCOM reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	10	DEC 17	COMPUAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	11	DEC 17	SOLIDSTATE reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	12	DEC 17	CERAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	13	DEC 17	THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
NEWS	14	DEC 30	EPFULL: New patent full text database to be available on STN
NEWS	15	DEC 30	CAPLUS - PATENT COVERAGE EXPANDED
NEWS	16	JAN 03	No connect-hour charges in EPFULL during January and February 2005
NEWS	17	FEB 25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	18	FEB 10	STN Patent Forums to be held in March 2005
NEWS	19	FEB 16	STN User Update to be held in conjunction with the 229th ACS National Meeting on March 13, 2005
NEWS	20	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	21	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	22	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	23	MAR 02	GBFULL: New full-text patent database on STN
NEWS	24	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	25	MAR 03	MEDLINE file segment of TOXCENTER reloaded

NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
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* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 13:14:48 ON 16 MAR 2005

=> file medline, uspatful, biosis, fsta, jicst, biotechds, wpids
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 1.68 1.68

FILE 'MEDLINE' ENTERED AT 13:19:26 ON 16 MAR 2005

FILE 'USPATFULL' ENTERED AT 13:19:26 ON 16 MAR 2005
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FILE 'WPIDS' ENTERED AT 13:19:26 ON 16 MAR 2005
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=> s cardiovascular disease
L1 298015 CARDIOVASCULAR DISEASE

=> s l1 and treatment
L2 61034 L1 AND TREATMENT

=> s LDL
L3 93918 LDL

=> sCRP
SCRP IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s CRP
L4 23399 CRP

=> s l4 and l3
L5 876 L4 AND L3

=> s l5 and l1
L6 240 L5 AND L1

=> s VLDL
L7 25858 VLDL

=> s l7 and l6
L8 34 L7 AND L6

=> s lactoferrin
L9 13291 LACTOFERRIN

=> s l8 and l9
L10 3 L8 AND L9

=> d l10 ti abs ibib tot

L10 ANSWER 1 OF 3 USPATFULL on STN
TI **Lactoferrin** in the reduction of circulating cholesterol, vascular inflammation, atherosclerosis and **cardiovascular disease**
AB The present invention relates to methods of using **lactoferrin** (LF) to reduce circulating levels of cholesterol and vascular inflammation, in order to treat, prevent or reduce the incidence of atherosclerosis and **cardiovascular disease**.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2004:197318 USPATFULL			
TITLE:	**Lactoferrin** in the reduction of circulating cholesterol, vascular inflammation, atherosclerosis and **cardiovascular disease**			
INVENTOR(S):	Varadhachary, Atul, Houston, TX, UNITED STATES Glynn, Peter, Houston, TX, UNITED STATES Wang, Yenyun, Houston, TX, UNITED STATES Engelmayer, Jose, Houston, TX, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004152623	A1	20040805	
APPLICATION INFO.:	US 2003-728275	A1	20031204	(10)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2002-430867P		20021204	(60)
	US 2003-498337P		20030827	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	FULBRIGHT & JAWORSKI, LLP, 1301 MCKINNEY, SUITE 5100, HOUSTON, TX, 77010-3095			
NUMBER OF CLAIMS:	34			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	5 Drawing Page(s)			
LINE COUNT:	1264			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 2 OF 3 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Treating a **cardiovascular disease** comprises administering to a subject an effective amount of a **lactoferrin** composition to provide an improvement in the **cardiovascular disease** in the subject;
involving vector-mediated gene transfer and expression in host cell for use in gene therapy
AN 2004-16843 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Treating a **cardiovascular disease** comprises administering to a subject an effective amount of a **lactoferrin** composition to provide an improvement in the **cardiovascular disease** in the subject.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method of modulating atherosclerosis in a subject comprising administering to the subject an effective amount of a **lactoferrin** composition to modulate atherosclerosis in the subject.
BIOTECHNOLOGY - Preferred Method: In treating a **cardiovascular disease**, the **cardiovascular disease** is atherosclerosis. The **lactoferrin** composition reduces levels of circulating total cholesterol, low-density lipoproteins (**LDL**), very low-density lipoproteins (**VLDL**), or triglycerides in the subject. The **lactoferrin** composition increases the levels of circulating high-density lipoproteins (HDL) in the subject. The **lactoferrin** composition reduces the levels of vascular inflammation, circulating C-reactive protein (**CRP**), proliferation of vascular smooth muscle cells, vascular spasm or vascular hyper-reactivity in the subject. The **lactoferrin** composition promotes endothelial integrity or healing in the subject. The

lactoferrin composition is dispersed in a carrier. The **lactoferrin** is mammalian **lactoferrin**. The **lactoferrin** is human or bovine. The **lactoferrin** is recombinant **lactoferrin**. The **lactoferrin** composition comprises an N-terminal **lactoferrin** variant. The N-terminal **lactoferrin** variant lacks at least the N-terminal glycine residue. The N-terminal **lactoferrin** variant comprises at least 1% to at least 50% of the **lactoferrin** composition. The **lactoferrin** composition reduces the production or activity of pro-inflammatory cytokines. The method further comprises administering a **lactoferrin** composition in combination with an anti-cholesterol agent or an anti-inflammatory agent. The anti-cholesterol agent is selected from cholesterol absorption inhibitors, bile acid sequestrants, nicotinic acid, fibric acids and HMG-coA reductase inhibitors. The bile acid sequestrants are selected from cholestyramine, colestipol and colesevalam. The fibric acids are selected from gemfibrozil, fenofibrate and clofibrate. The HMG-coA reductase inhibitors are selected from lovastatin, pravastatin, simvastatin, fluvastatin, atorvastatin and cerivastatin. In modulating atherosclerosis in a subject, the modulating is reducing the incidence or severity of atherosclerosis in the subject.

ACTIVITY - Cardiant; Antiarteriosclerotic. No biological data given.

MECHANISM OF ACTION - Gene therapy; HMG-coA reductase inhibitor.

USE - The method is useful for treating a **cardiovascular disease**, e.g. atherosclerosis (claimed).

ADMINISTRATION - Dosage is 1 ng-20 g per day or 0.1-5 g per day. The **lactoferrin** composition is administered parenterally, e.g. subcutaneously, intramuscularly, intraperitoneally, intravenously, intraarterially, intramyocardially, transendocardially, transepicardially, or intrathecally, or orally (all claimed). (38 pages)

ACCESSION NUMBER: 2004-16843 BIOTECHDS
TITLE: Treating a **cardiovascular disease** comprises administering to a subject an effective amount of a **lactoferrin** composition to provide an improvement in the **cardiovascular disease** in the subject

;

involving vector-mediated gene transfer and expression in host cell for use in gene therapy

AUTHOR: VARADHACHARY A; GLYNN P; WANG Y; ENGELMAYER J
PATENT ASSIGNEE: AGENNIX INC; VARADHACHARY A
PATENT INFO: WO 2004050037 17 Jun 2004
APPLICATION INFO: WO 2003-US38540 4 Dec 2003
PRIORITY INFO: US 2003-498337 27 Aug 2003; US 2002-430867 4 Dec 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2004-460986 [43]

L10 ANSWER 3 OF 3 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treating a **cardiovascular disease** comprises administering to a subject an effective amount of a **lactoferrin** composition to provide an improvement in the **cardiovascular disease** in the subject.
AN 2004-460986 [43] WPIDS
AB WO2004050037 A UPAB: 20040709

NOVELTY - Treating a **cardiovascular disease** comprises administering to a subject an effective amount of a **lactoferrin** composition to provide an improvement in the **cardiovascular disease** in the subject.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method of modulating atherosclerosis in a subject comprising administering to the subject an effective amount of a **lactoferrin** composition to modulate atherosclerosis in the subject.

ACTIVITY - Cardiant; Antiarteriosclerotic. No biological data given.

MECHANISM OF ACTION - Gene therapy; HMG-coA reductase inhibitor.

USE - The method is useful for treating a **cardiovascular disease**, e.g. atherosclerosis (claimed).

Dwg.0/5

ACCESSION NUMBER: 2004-460986 [43] WPIDS
DOC. NO. CPI: C2004-172138

TITLE: Treating a **cardiovascular disease** comprises administering to a subject an effective amount of a **lactoferrin** composition to provide an improvement in the **cardiovascular disease** in the subject.
DERWENT CLASS: B04 D16
INVENTOR(S): ENGELMAYER, J; GLYNN, P; VARADHACHARY, A; WANG, Y
PATENT ASSIGNEE(S): (ENGE-I) ENGELMAYER J; (GLYN-I) GLYNN P; (VARA-I) VARADHACHARY A; (WANG-I) WANG Y; (AGEN-N) AGENNIX INC
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004050037	A2	20040617	(200443)*	EN	38
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2004152623	A1	20040805	(200452)		
AU 2003291206	A1	20040623	(200472)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004050037	A2	WO 2003-US38540	20031204
US 2004152623	A1 Provisional	US 2002-430867P	20021204
	Provisional	US 2003-498337P	20030827
		US 2003-728275	20031204
AU 2003291206	A1	AU 2003-291206	20031204

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003291206	A1 Based on	WO 2004050037

PRIORITY APPLN. INFO: US 2003-498337P 20030827; US 2002-430867P 20021204; US 2003-728275 20031204

```
=> s cholestryramine
L11          83 CHOLESTRYRAMINE

=> s cholestipol
L12         105 CHOLESTIPOL

=> s l11 and l12
L13           1 L11 AND L12

=> d l13 ti abs ibib tot

L13  ANSWER 1 OF 1  USPATFULL on STN
TI     Lactoferrin in the reduction of circulating cholesterol, vascular
       inflammation, atherosclerosis and cardiovascular disease
AB     The present invention relates to methods of using lactoferrin (LF) to
       reduce circulating levels of cholesterol and vascular inflammation, in
       order to treat, prevent or reduce the incidence of atherosclerosis and
       cardiovascular disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:       2004:197318  USPATFULL
TITLE:                  Lactoferrin in the reduction of circulating
```

cholesterol, vascular inflammation, atherosclerosis and cardiovascular disease

INVENTOR(S): Varadhachary, Atul, Houston, TX, UNITED STATES
Glynn, Peter, Houston, TX, UNITED STATES
Wang, Yenyun, Houston, TX, UNITED STATES
Engelmayer, Jose, Houston, TX, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004152623	A1	20040805	
APPLICATION INFO.:	US 2003-728275	A1	20031204	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2002-430867P	20021204	(60)
	US 2003-498337P	20030827	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FULBRIGHT & JAWORSKI, LLP, 1301 MCKINNEY, SUITE 5100, HOUSTON, TX, 77010-3095
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 1264
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> s atherosclerosis
L14 138829 ATHEROSCLEROSIS

=> s l14 and lactoferrin
L15 175 L14 AND LACTOFERRIN

=> s l15 and treatment
L16 154 L15 AND TREATMENT

=> s l16 and (reduced incidence)
4 FILES SEARCHED...
L17 4 L16 AND (REDUCED INCIDENCE)

=> d l17 ti abs ibib tot

L17 ANSWER 1 OF 4 USPATFULL on STN
TI Proteins, polynucleotides encoding them and methods of using the same
AB Disclosed herein are nucleic acid sequences that encode novel polypeptides. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivatives, variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, **treatment**, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2004:7324 USPATFULL
TITLE: Proteins, polynucleotides encoding them and methods of using the same

INVENTOR(S): Padigaru, Muralidhara, Branford, CT, UNITED STATES
Alsobrook, John P., II, Madison, CT, UNITED STATES
Colman, Steven D., Guilford, CT, UNITED STATES
Spytek, Kimberly A., New Haven, CT, UNITED STATES
Boldog, Ferenc L., North Haven, CT, UNITED STATES
Vernet, Corine A.M., Branford, CT, UNITED STATES
Li, Li, Branford, CT, UNITED STATES
Shenoy, Suresh G., Branford, CT, UNITED STATES
Casman, Stacie J., North Haven, CT, UNITED STATES
Guo, Xiaojia (Sasha), Branford, CT, UNITED STATES
Edinger, Shlomit R., New Haven, CT, UNITED STATES

MacDougall, John R., Hamden, CT, UNITED STATES
Malyankar, Uriel M., Branford, CT, UNITED STATES
Patturajan, Meera, Branford, CT, UNITED STATES
Shimkets, Richard A., Guilford, CT, UNITED STATES
Pena, Carol E. A., New Haven, CT, UNITED STATES
Tchernev, Velizar T., Branford, CT, UNITED STATES
Zerhusen, Bryan D., Branford, CT, UNITED STATES
Millet, Isabelle, Milford, CT, UNITED STATES
Miller, Charles E., Guilford, CT, UNITED STATES
Lepley, Denise M., Branford, CT, UNITED STATES
Smithson, Glennda, Guilford, CT, UNITED STATES
Baumgartner, Jason C., New Haven, CT, UNITED STATES
Herrmann, John L., Guilford, CT, UNITED STATES
Peyman, John A., New Haven, CT, UNITED STATES
Gorman, Linda, Branford, CT, UNITED STATES
Mezes, Peter D., Old Lyme, CT, UNITED STATES
Kekuda, Ramesh, Norwalk, CT, UNITED STATES
Taupier, Raymond J., JR., East Haven, CT, UNITED STATES
Gerlach, Valerie, Branford, CT, UNITED STATES
Grosse, William M., Branford, CT, UNITED STATES
Liu, Xiaohong, Lexington, MA, UNITED STATES
Ellerman, Karen, Branford, CT, UNITED STATES
Rothenberg, Mark, Clinton, CT, UNITED STATES
Stone, David J., Guilford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004005557	A1	20040108	
APPLICATION INFO.:	US 2002-51874	A1	20020116	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-261376P	20010116	(60)
	US 2001-268595P	20010214	(60)
	US 2001-325306P	20010927	(60)
	US 2001-262587P	20010118	(60)
	US 2001-272409P	20010228	(60)
	US 2001-262454P	20010118	(60)
	US 2001-276777P	20010316	(60)
	US 2001-291672P	20010517	(60)
	US 2001-330336P	20011018	(60)
	US 2001-265530P	20010131	(60)
	US 2001-345202P	20011109	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MINTZ, LEVIN, COHN, FERRIS,, GLOVSKY and POPEO, P.C., One Financial Center, Boston, MA, 02111
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
LINE COUNT: 16208
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 2 OF 4 USPATFULL on STN
TI IGF-binding protein-derived peptide or small molecule
AB New compositions based on IGF-binding protein sequences are provided. New tools for high-throughput research are provided. New methods for the **treatment** of human disease are provided. IGFBP-3-derived peptide or small molecule is administered to subjects having disease, thereby alleviating the symptoms of the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:319241 USPATFULL
TITLE: IGF-binding protein-derived peptide or small molecule
INVENTOR(S): Mascarenhas, Desmond, Los Altos Hills, CA, UNITED STATES

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 2003224990	A1	20031204
APPLICATION INFO.:	US 2003-383999	A1	20030307 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-264672, filed on 4 Oct 2002, PENDING Continuation-in-part of Ser. No. US 2002-215759, filed on 9 Aug 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-323267P	20010918 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Nicholas S. Buffinger, Morrison & Foerster LLP, 755 Page Mill Road, Palo Alto, CA, 94304-1018
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 24 Drawing Page(s)
LINE COUNT: 2168
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 3 OF 4 USPATFULL on STN
TI IGF-binding protein-derived peptide or small molecule
AB New compositions based on IGF-binding protein sequences are provided. New tools for high-throughput research are provided. New methods for the **treatment** of human disease are provided. IGFBP-3-derived peptide or small molecule is administered to subjects having disease, thereby alleviating the symptoms of the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:231631 USPATFULL
TITLE: IGF-binding protein-derived peptide or small molecule
INVENTOR(S): Mascarenhas, Desmond, Los Altos Hills, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003161829	A1	20030828
APPLICATION INFO.:	US 2002-264672	A1	20021004 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-215759, filed on 9 Aug 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-323267P	20010918 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Nicholas S. Buffinger, Morrison & Foerster LLP, 755 Page Mill Road, Palo Alto, CA, 94304-1018
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 2061
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 4 OF 4 USPATFULL on STN
TI Blood cell deficiency **treatment** method
AB The invention relates to the use of compounds to treat a number of conditions, such as thrombocytopenia, neutropenia or the delayed effects of radiation therapy. Compounds that can be used in the invention include methyl-2,3,4-trihydroxy-1-O-(7,17-dioxoandrost-5-ene-3β-yl)-β-D-glucopyranosiduronate, 16α,3α-dihydroxy-5α-androstan-17-one or 3,7,16,17-tetrahydroxyandrost-5-ene, 3,7,16,17-tetrahydroxyandrost-4-ene,3,7,16,17-tetrahydroxyandrost-1-ene or 3,7,16,17-tetrahydroxyandrostane that can be used in the **treatment** method.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2003:120747 USPATFULL

TITLE: Blood cell deficiency **treatment** method

INVENTOR(S): Ahlem, Clarence N., San Diego, CA, UNITED STATES
Reading, Christopher, San Diego, CA, UNITED STATES
Frincke, James, San Diego, CA, UNITED STATES
Stickney, Dwight, Granite Bay, CA, UNITED STATES
Lardy, Henry A., Madison, WI, UNITED STATES
Marwah, Padma, Middleton, WI, UNITED STATES
Marwah, Ashok, Middleton, WI, UNITED STATES
Prendergast, Patrick T., Straffan, IRELAND

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003083231	A1	20030501
APPLICATION INFO.:	US 2002-87929	A1	20020301 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-675470, filed on 28 Sep 2000, PENDING Continuation-in-part of Ser. No. US 2001-820483, filed on 29 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2000-535675, filed on 23 Mar 2000, PENDING Continuation-in-part of Ser. No. US 1999-449004, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449184, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449042, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-461026, filed on 15 Dec 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-586673, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-586672, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 1999-414905, filed on 8 Oct 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-161453P	19991025 (60)
	US 2001-272624P	20010301 (60)
	US 2001-323016P	20010911 (60)
	US 2001-340045P	20011130 (60)
	US 2001-328738P	20011011 (60)
	US 2001-338015P	20011108 (60)
	US 2001-343523P	20011220 (60)
	US 1999-126056P	19991019 (60)
	US 1999-124087P	19990311 (60)
	US 1998-109923P	19981124 (60)
	US 1998-109924P	19981124 (60)
	US 1998-110127P	19981127 (60)
	US 1998-112206P	19981215 (60)
	US 1999-145823P	19990727 (60)
	US 1999-137745P	19990603 (60)
	US 1999-140028P	19990616 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
LINE COUNT: 19428
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

```
=> s cardiovascular disease and lactoferrin
L18         88 CARDIOVASCULAR DISEASE AND LACTOFERRIN

=> s l18 and treatment
L19         72 L18 AND TREATMENT

=> s l19 and (reduced incidence)
L20          3 L19 AND (REDUCED INCIDENCE)

=> d l20 ti abs ibib ott
```

'OTT' IS NOT A VALID FORMAT FOR FILE 'USPATFULL'

The following are valid formats:

The default display format is STD.

ABS -------- AB
ALL -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
EXF, ARTU
ALLG ------- ALL plus PAGE.DRAW
BIB -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI,
PRAI, DT, FS, EXNAM, LREP, CLMN, ECL, DRWN, LN.CNT
BIB.EX ----- BIB for original and latest publication
BIBG ------- BIB plus PAGE.DRAW
BROWSE ----- See "HELP BROWSE" or "HELP DISPLAY BROWSE". BROWSE must
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CAS -------- OS, CC, SX, ST, IT
CBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PRAI, DT, FS
DALL ------- ALL, delimited for post-processing
FP --------- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI,
PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL,
NCLM, NCLS, EXF, REP, REN, ARTU, EXNAM, LREP,
CLMN, DRWN, AB
FP.EX ------ FP for original and latest publication
FPALL ------ PI, TI, IN, INA, PA, PAA, PAT, PETRM, DCD, AI,
RLI, PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL, NCLM,
NCLS, EXF, REP, REN,ARTU, EXNAM, LREP, CLMN, DRWN, AB,
PARN, SUMM, DRWD, DETD, CLM
FPBIB ------ PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI,
RLI, PRAI, REP, REN, EXNAM, LREP, CLM, CLMN, DRWN
FHITSTR ---- HIT RN, its text modification, its CA index name, and
its structure diagram
FPG -------- FP plus PAGE.DRAW
GI --------- PN and page image numbers
HIT -------- All fields containing hit terms
HITRN ------ HIT RN and its text modification
HITSTR ----- HIT RN, its text modification, its CA index name, and
its structure diagram
IABS ------- ABS, indented with text labels
IALL ------- ALL, indented with text labels
IALLG ------ IALL plus PAGE.DRAW
IBIB ------- BIB, indented with text labels
IBIB.EX ---- IBIB for original and latest publication
IBIBG ------ IBIB plus PAGE.DRAW
IMAX ------- MAX, indented with text labels
IMAX.EX ---- IMAX for original and latest publication
IND -------- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
EXF, ARTU, OS, CC, SX, ST, IT
ISTD ------- STD, indented with text labels
KWIC ------- All hit terms plus 20 words on either side
MAX -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
EXF, ARTU OS, CC, SX, ST, IT
MAX.EX ----- MAX for original and latest publication
OCC -------- List of display fields containing hit terms
SBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
DT, FS, LN.CNT
SCAN ------- AN, TI, NCL, NCLM, NCLS, IC, ICM, ICS (random display
without answer number. SCAN must be entered on the
same line as DISPLAY, e.g., D SCAN)
STD -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
DT, FS, LN.CNT, INCL, INCLM, INCLS, NCL, NCLM, NCLS,
IC, ICM, ICS, EXF (STD is the default)

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STD.EX ----- STD for original and latest publication
TRIAL ------ AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC,
             ICM, ICS

ENTER DISPLAY FORMAT (STD):end

=> d his

     (FILE 'HOME' ENTERED AT 13:14:48 ON 16 MAR 2005)

     FILE 'MEDLINE, USPATFULL, BIOSIS, FSTA, JICST-EPLUS, BIOTECHDS, WPIDS'
     ENTERED AT 13:19:26 ON 16 MAR 2005
L1        298015 S CARDIOVASCULAR DISEASE
L2         61034 S L1 AND TREATMENT
L3         93918 S LDL
L4         23399 S CRP
L5           876 S L4 AND L3
L6           240 S L5 AND L1
L7         25858 S VLDL
L8            34 S L7 AND L6
L9         13291 S LACTOFERRIN
L10            3 S L8 AND L9
L11           83 S CHOLESTRYRAMINE
L12          105 S CHOLESTIPOL
L13            1 S L11 AND L12
L14       138829 S ATHEROSCLEROSIS
L15          175 S L14 AND LACTOFERRIN
L16          154 S L15 AND TREATMENT
L17            4 S L16 AND (REDUCED INCIDENCE)
L18           88 S CARDIOVASCULAR DISEASE AND LACTOFERRIN
L19           72 S L18 AND TREATMENT
L20            3 S L19 AND (REDUCED INCIDENCE)

=> d l20 ti abs ibib tot

L20  ANSWER 1 OF 3  USPATFULL on STN
TI     IGF-binding protein-derived peptide or small molecule
AB     New compositions based on IGF-binding protein sequences are provided.
       New tools for high-throughput research are provided. New methods for the
       treatment of human disease are provided. IGFBP-3-derived peptide
       or small molecule is administered to subjects having disease, thereby
       alleviating the symptoms of the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:        2003:319241  USPATFULL
TITLE:                   IGF-binding protein-derived peptide or small molecule
INVENTOR(S):             Mascarenhas, Desmond, Los Altos Hills, CA, UNITED
                         STATES

                              NUMBER       KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2003224990      A1   20031204
APPLICATION INFO.:       US 2003-383999     A1   20030307  (10)
RELATED APPLN. INFO.:    Continuation-in-part of Ser. No. US 2002-264672, filed
                         on 4 Oct 2002, PENDING Continuation-in-part of Ser. No.
                         US 2002-215759, filed on 9 Aug 2002, PENDING

                                NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 2001-323267P    20010918 (60)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    Nicholas S. Buffinger, Morrison & Foerster LLP, 755
                         Page Mill Road, Palo Alto, CA, 94304-1018
NUMBER OF CLAIMS:        34
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      24 Drawing Page(s)
LINE COUNT:              2168
```

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L20 ANSWER 2 OF 3 USPATFULL on STN
TI IGF-binding protein-derived peptide or small molecule
AB New compositions based on IGF-binding protein sequences are provided. New tools for high-throughput research are provided. New methods for the **treatment** of human disease are provided. IGFBP-3-derived peptide or small molecule is administered to subjects having disease, thereby alleviating the symptoms of the disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:231631 USPATFULL		
TITLE:	IGF-binding protein-derived peptide or small molecule		
INVENTOR(S):	Mascarenhas, Desmond, Los Altos Hills, CA, UNITED STATES		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003161829	A1	20030828
APPLICATION INFO.:	US 2002-264672	A1	20021004 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2002-215759, filed on 9 Aug 2002, PENDING		
	NUMBER	DATE	
PRIORITY INFORMATION:	US 2001-323267P	20010918 (60)	
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	Nicholas S. Buffinger, Morrison & Foerster LLP, 755 Page Mill Road, Palo Alto, CA, 94304-1018		
NUMBER OF CLAIMS:	30		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	16 Drawing Page(s)		
LINE COUNT:	2061		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L20 ANSWER 3 OF 3 USPATFULL on STN
TI Blood cell deficiency **treatment** method
AB The invention relates to the use of compounds to treat a number of conditions, such as thrombocytopenia, neutropenia or the delayed effects of radiation therapy. Compounds that can be used in the invention include methyl-2,3,4-trihydroxy-1-O-(7,17-dioxoandrost-5-ene-3β-yl)-β-D-glucopyranosiduronate, 16α,3α-dihydroxy-5α-androstan-17-one or 3,7,16,17-tetrahydroxyandrost-5-ene, 3,7,16,17-tetrahydroxyandrost-4-ene,3,7,16,17-tetrahydroxyandrost-1-ene or 3,7,16,17-tetrahydroxyandrostane that can be used in the **treatment** method.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2003:120747 USPATFULL		
TITLE:	Blood cell deficiency **treatment** method		
INVENTOR(S):	Ahlem, Clarence N., San Diego, CA, UNITED STATES Reading, Christopher, San Diego, CA, UNITED STATES Frincke, James, San Diego, CA, UNITED STATES Stickney, Dwight, Granite Bay, CA, UNITED STATES Lardy, Henry A., Madison, WI, UNITED STATES Marwah, Padma, Middleton, WI, UNITED STATES Marwah, Ashok, Middleton, WI, UNITED STATES Prendergast, Patrick T., Straffan, IRELAND		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003083231	A1	20030501
APPLICATION INFO.:	US 2002-87929	A1	20020301 (10)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-675470, filed on 28 Sep 2000, PENDING Continuation-in-part of Ser. No. US 2001-820483, filed on 29 Mar 2001, PENDING Continuation-in-part of Ser. No. US 2000-535675, filed		

on 23 Mar 2000, PENDING Continuation-in-part of Ser. No. US 1999-449004, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449184, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-449042, filed on 24 Nov 1999, ABANDONED Continuation-in-part of Ser. No. US 1999-461026, filed on 15 Dec 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-586673, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-586672, filed on 1 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 1999-414905, filed on 8 Oct 1999, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-161453P	19991025 (60)
	US 2001-272624P	20010301 (60)
	US 2001-323016P	20010911 (60)
	US 2001-340045P	20011130 (60)
	US 2001-328738P	20011011 (60)
	US 2001-338015P	20011108 (60)
	US 2001-343523P	20011220 (60)
	US 1999-126056P	19991019 (60)
	US 1999-124087P	19990311 (60)
	US 1998-109923P	19981124 (60)
	US 1998-109924P	19981124 (60)
	US 1998-110127P	19981127 (60)
	US 1998-112206P	19981215 (60)
	US 1999-145823P	19990727 (60)
	US 1999-137745P	19990603 (60)
	US 1999-140028P	19990616 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOLLIS-EDEN PHARMACEUTICALS, INC., 4435 EASTGATE MALL, SUITE 400, SAN DIEGO, CA, 92121
NUMBER OF CLAIMS: 45
EXEMPLARY CLAIM: 1
LINE COUNT: 19428
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 5 of 5 returned.

☐ 1. Document ID: US 6140552 A

L1: Entry 1 of 5 File: USPT Oct 31, 2000

US-PAT-NO: 6140552
DOCUMENT-IDENTIFIER: US 6140552 A

TITLE: Production of recombinant polypeptides by bovine species and transgenic methods

DATE-ISSUED: October 31, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deboer; Herman A.	Roelofarendsyeen			NL
Strijker; Rein	Oegstgeest			NL
Heyneker; Herbert L.	Hillsborough	CA		
Platenburg; Gerard	Voorschoten			NL
Lee; Sang He	Leiden			NL
Pieper; Frank	Utrecht			NL
Krimpenfort; Paul J. A.	Heemstede			NL

US-CL-CURRENT: 800/15; 800/3, 800/4, 800/7, 800/8

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 6066725 A

L1: Entry 2 of 5 File: USPT May 23, 2000

US-PAT-NO: 6066725
DOCUMENT-IDENTIFIER: US 6066725 A

TITLE: Production of recombinant polypeptides by bovine species and transgenic methods

DATE-ISSUED: May 23, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
DeBoer; Herman A.	Roelofarendsveen			NL
Strijker; Rein	Oegstgeest			NL
Heyneker; Herbert L.	Hillsborough	CA		
Platenburg; Gerard	Voorschoten			NL
Lee; Sang He	Leiden			NL
Pieper; Frank	Utrecht			NL
Krimpenfort; Paul J. A.	Heemstede			NL

US-CL-CURRENT: 536/23.5; 435/69.1, 536/23.1

☐ 3. Document ID: US 6013857 A

L1: Entry 3 of 5 File: USPT Jan 11, 2000

US-PAT-NO: 6013857
DOCUMENT-IDENTIFIER: US 6013857 A

TITLE: Transgenic bovines and milk from transgenic bovines

DATE-ISSUED: January 11, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deboer; Herman A.	Roelofarendsveen			NL
Strijker; Rein	Oegstgeest			NL
Heyneker; Herbert L.	Hillsborough	CA		
Platenburg; Gerard	Voorschoten			NL
Lee; Sang He	Leiden			NL
Pieper; Frank	Utrecht			NL
Krimpenfort; Paul J. A.	Heemstede			NL

US-CL-CURRENT: 800/15; 424/535, 426/556, 426/580, 426/587, 426/588, 435/3, 435/69.1, 800/13, 800/4, 800/5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

☐ 4. Document ID: US 5741957 A

L1: Entry 4 of 5 File: USPT Apr 21, 1998

US-PAT-NO: 5741957
DOCUMENT-IDENTIFIER: US 5741957 A

TITLE: Transgenic bovine

DATE-ISSUED: April 21, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deboer; Herman A.	Roelofarendsveen			NL
Strijker; Rein	Oegstgeest			NL
Heyneker; Herbert L.	Hillsborough	CA		
Platenburg; Gerard	Voorschoten			NL
Lee; Sang He	Leiden			NL
Pieper; Frank	Utrecht			NL
Krimpenfort; Paul J. A.	Heemstede			NL

US-CL-CURRENT: 800/7; 435/69.1, 800/15, 800/25

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

☐ 5. Document ID: US 5633076 A

US-PAT-NO: 5633076
DOCUMENT-IDENTIFIER: US 5633076 A

TITLE: Method of producing a transgenic bovine or transgenic bovine embryo

DATE-ISSUED: May 27, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
DeBoer; Herman A.	Roelofarendsveen			NL
Strijker; Rein	Oegstgeest			NL
Heyneker; Herbert L.	Hillsborough	CA		
Platenburg; Gerard	Voorschoten			NL
Lee; Sang H.	Leiden			NL
Pieper; Frank	Utrecht			NL
Krimpenfort; Paul J. A.	Heemstede			NL

US-CL-CURRENT: 800/25

Terms	Documents
lactoferrin adj2 cholesterol	5

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 8 of 8 returned.

☐ 1. Document ID: US 4491616 A

L6: Entry 1 of 8 File: USPT Jan 1, 1985

US-PAT-NO: 4491616
DOCUMENT-IDENTIFIER: US 4491616 A

TITLE: Resinous polymer sheet material having surface decorative effects of contrasting gloss and method of making the same

DATE-ISSUED: January 1, 1985

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Schmidle; Claude J.	Trenton	NJ		
Varadhachary; Seevaram N.	Newtown	PA		

US-CL-CURRENT: 428/158; 427/272, 427/280, 427/494, 427/510, 428/159, 428/201, 428/212, 428/308.4, 428/913

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 4389514 A

L6: Entry 2 of 8 File: USPT Jun 21, 1983

US-PAT-NO: 4389514
DOCUMENT-IDENTIFIER: US 4389514 A

TITLE: Accelerated polymerization of acrylic monomers initiated by dialkyl and diaralkyl peroxide free radical generators in the presence of tin accelerators

DATE-ISSUED: June 21, 1983

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Schmidle; Claude J.	Trenton	NJ		
Varadhachary; Seevaram N.	Newtown	PA		

US-CL-CURRENT: 525/364; 525/370, 526/184, 526/192

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 3. Document ID: US 4361626 A

L6: Entry 3 of 8 File: USPT Nov 30, 1982

US-PAT-NO: 4361626
DOCUMENT-IDENTIFIER: US 4361626 A

TITLE: Methods for bonding dissimilar synthetic polymeric materials and the products involved in and resulting from such methods

DATE-ISSUED: November 30, 1982

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Boba; Joseph	Fort Lee	NJ		
Varadhachary; Seevaram N.	Newtown	PA		
Pogozelski; Vincent F.	Newtown	PA		

US-CL-CURRENT: 428/420; 427/302, 427/333, 427/372.2, 427/520, 428/423.3, 428/424.4, 428/424.6, 428/424.8, 428/522, 522/126, 522/95, 522/96

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

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☐ 4. Document ID: US 4337296 A

L6: Entry 4 of 8 File: USPT Jun 29, 1982

US-PAT-NO: 4337296
DOCUMENT-IDENTIFIER: US 4337296 A

TITLE: Methods for bonding dissimilar synthetic polymeric materials and the products involved in and resulting from such methods

DATE-ISSUED: June 29, 1982

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Varadhachary; Seevaram N.	Newtown	PA		

US-CL-CURRENT: 428/420; 427/302, 427/333, 427/372.2, 427/517, 427/520, 428/423.3, 428/424.4, 428/424.6, 428/424.8, 428/522, 522/111, 522/95, 522/96

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

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☐ 5. Document ID: US 4333987 A

L6: Entry 5 of 8 File: USPT Jun 8, 1982

US-PAT-NO: 4333987
DOCUMENT-IDENTIFIER: US 4333987 A

TITLE: Methods for bonding dissimilar synthetic polymeric materials and the products involved in and resulting from such methods

DATE-ISSUED: June 8, 1982

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kwart; Harold	Newark	DE	19711	
Varadhachary; Seevaram N.	Newtown	PA	18940	

US-CL-CURRENT: 428/419; 427/372.2, 427/520, 428/420, 428/424.6, 522/20, 522/66, 522/96, 522/97

☐ 6. Document ID: US 4273819 A

L6: Entry 6 of 8 File: USPT Jun 16, 1981

US-PAT-NO: 4273819
DOCUMENT-IDENTIFIER: US 4273819 A

TITLE: Differential gloss products and methods of making the same

DATE-ISSUED: June 16, 1981

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Schmidle; Claude J.	Trenton	NJ		
Varadhachary; Seevaram N.	Newtown	PA		

US-CL-CURRENT: 428/159; 156/219, 156/220, 156/240, 156/246, 156/247, 156/277, 156/79, 264/52, 264/DIG.82, 427/264, 427/373, 428/207, 428/304.4

☐ 7. Document ID: US 3966857 A

L6: Entry 7 of 8 File: USPT Jun 29, 1976

US-PAT-NO: 3966857
DOCUMENT-IDENTIFIER: US 3966857 A

TITLE: Lubrication of extruded materials

DATE-ISSUED: June 29, 1976

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Charlton; Ralph W.	Newfoundland	NJ		
Varadhachary; Seevaram N.	North Plainfield	NJ		

US-CL-CURRENT: 264/75; 264/176.1, 264/211, 264/245, 264/349

☐ 8. Document ID: US 3787280 A

L6: Entry 8 of 8 File: USPT Jan 22, 1974

US-PAT-NO: 3787280
DOCUMENT-IDENTIFIER: US 3787280 A

TITLE: RESINOUS PRODUCT HAVING SHARP COLOR DEFINITIONS THEREIN

DATE-ISSUED: January 22, 1974

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY

Conger; Robert P.	Park Ridge	NJ
Varadhachary; Seevaram N.	North Plainfield	NJ

US-CL-CURRENT: 525/82; 156/62.2, 264/245, 264/73, 525/305

Terms	Documents
varadhachary.in.	8

Hit List

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Search Results - Record(s) 1 through 10 of 12 returned.

☐ 1. Document ID: US 6743908 B2

L11: Entry 1 of 12 File: USPT Jun 1, 2004

US-PAT-NO: 6743908
DOCUMENT-IDENTIFIER: US 6743908 B2

TITLE: Single-chain antigen-binding proteins capable of glycosylation, production and uses thereof

DATE-ISSUED: June 1, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Filpula; David	Piscataway	NJ		
Wang; Maoliang	E. Brunswick	NJ		
Shorr; Robert	Edison	NJ		
Whitlow; Marc	El Sobrante	CA		
Lee; Lihsyng S.	Princeton Junction	NJ		

US-CL-CURRENT: 536/23.53; 435/320.1, 435/69.1, 435/69.6

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 6743896 B2

L11: Entry 2 of 12 File: USPT Jun 1, 2004

US-PAT-NO: 6743896
DOCUMENT-IDENTIFIER: US 6743896 B2

TITLE: Single-chain antigen-binding proteins capable of glycosylation, production and uses thereof

DATE-ISSUED: June 1, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Filpula; David	Piscataway	NJ		
Wang; Maoliang	E. Brunswick	NJ		
Shorr; Robert	Edison	NJ		
Whitlow; Marc	El Sobrante	CA		
Lee; Lihsyng S.	Princeton Junction	NJ		

US-CL-CURRENT: 530/387.3; 435/188, 530/391.1, 530/391.7

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

☐ 3. Document ID: US 6323322 B1

L11: Entry 3 of 12 File: USPT Nov 27, 2001

US-PAT-NO: 6323322
DOCUMENT-IDENTIFIER: US 6323322 B1

TITLE: Single-chain antigen-binding proteins capable of glycosylation, production and uses thereof

DATE-ISSUED: November 27, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Filpula; David	Piscataway	NJ		
Wang; Maoliang	E. Brunswick	NJ		
Shorr; Robert	Edison	NJ		
Whitlow; Marc	El Sobrante	CA		
Lee; Lihsyng S.	Princeton Junction	NJ		

US-CL-CURRENT: 530/387.3; 530/391.3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

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☐ 4. Document ID: US 6197319 B1

L11: Entry 4 of 12 File: USPT Mar 6, 2001

US-PAT-NO: 6197319
DOCUMENT-IDENTIFIER: US 6197319 B1

TITLE: Cosmetic compositions containing polysaccharide/protein complexes

DATE-ISSUED: March 6, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Wang; Tian Xiang	Edison	NJ		
DiGirolamo; Debra Marsha Verdon	Holmdel	NJ		
Russ; Julio Gans	Westfield	NJ		

US-CL-CURRENT: 424/401; 424/59, 424/63, 424/70.1, 424/70.13, 424/70.14, 424/70.19, 424/70.27, 424/70.28, 424/74, 514/844 , 514/845, 514/846, 514/847

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Ima

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☐ 5. Document ID: US 6184371 B1

L11: Entry 5 of 12 File: USPT Feb 6, 2001

US-PAT-NO: 6184371
DOCUMENT-IDENTIFIER: US 6184371 B1

TITLE: Lactoferrin receptor genes of Moraxella

DATE-ISSUED: February 6, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Loosmore; Sheena M.	Aurora			CA
Du; Run-Pan	Thornhill			CA
Wang; Quijun	Thornhill			CA
Yang; Yan-Ping	Willowdale			CA
Klein; Michel H.	Willowdale			CA

US-CL-CURRENT: 536/23.7; 424/200.1, 424/251.1, 435/252.3, 435/320.1, 435/69.1, 435/69.3, 435/69.7, 536/23.1, 536/24.3, 536/24.32

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

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☐ 6. Document ID: US 6171586 B1

L11: Entry 6 of 12 File: USPT Jan 9, 2001

US-PAT-NO: 6171586
DOCUMENT-IDENTIFIER: US 6171586 B1

TITLE: Antibody formulation

DATE-ISSUED: January 9, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Lam; Xanthe M.	San Francisco	CA		
Oeswein; James Q.	Moss Beach	CA		
Ongpipattanakul; Boonsri	Bangkok			TH
Shahrokh; Zahra	San Francisco	CA		
Wang; Sharon X.	San Mateo	CA		
Weissburg; Robert P.	Greenville	DE		
Wong; Rita L.	San Mateo	CA		

US-CL-CURRENT: 424/130.1; 424/141.1, 424/152.1, 424/154.1, 424/173.1, 530/388.75

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

---

☐ 7. Document ID: US 6171581 B1

L11: Entry 7 of 12 File: USPT Jan 9, 2001

US-PAT-NO: 6171581
DOCUMENT-IDENTIFIER: US 6171581 B1

TITLE: Water and oil emulsion solid antiperspirant/deodorant compositions

DATE-ISSUED: January 9, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Joshi; Vijay Kumar	Livingston	NJ		
Shalotsky; Charles George	Chatham	NJ		
Wang; Tian Xiang	Edison	NJ		

US-CL-CURRENT: 424/65; 424/400, 424/401, 424/DIG.5, 514/937, 514/944

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

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☐ 8. Document ID: US 6042815 A

L11: Entry 8 of 12 File: USPT Mar 28, 2000

US-PAT-NO: 6042815
DOCUMENT-IDENTIFIER: US 6042815 A

TITLE: Water and oil emulsion solid cosmetic composition

DATE-ISSUED: March 28, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Kellner; David Martin	Hollis	NY		
Russ; Julio Gans	Westfield	NJ		
Sandewicz; Ida Marie	Spotswood	NJ		
Shandler; Robin Felice	Commack	NY		
Wang; Tian Xiang	Edison	NJ		

US-CL-CURRENT: 424/63; 424/400, 424/401, 424/59, 424/64, 424/65, 514/844, 514/937, 514/944

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

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☐ 9. Document ID: US 5977337 A

L11: Entry 9 of 12 File: USPT Nov 2, 1999

US-PAT-NO: 5977337
DOCUMENT-IDENTIFIER: US 5977337 A

TITLE: Lactoferrin receptor genes of Moraxella

DATE-ISSUED: November 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Loosmore; Sheena M.	Aurora			CA
Du; Run-Pan	Thornhill			CA
Wang; Quijun	Thornhill			CA
Yang; Yan-Ping	Willowdale			CA
Klein; Michel H.	Willowdale			CA

US-CL-CURRENT: 536/23.7; 424/256.1, 435/69.1, 435/69.3, 435/69.4, 530/350, 536/23.1, 536/24.3, 536/24.32

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

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☐ 10. Document ID: US 5837247 A

L11: Entry 10 of 12 File: USPT Nov 17, 1998

US-PAT-NO: 5837247
DOCUMENT-IDENTIFIER: US 5837247 A

TITLE: Chemotactic agents for t-cells

DATE-ISSUED: November 17, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Oppenhelm; Joost J.	Bethesda	MD		
Michiel; Dennis	Funkstown	MD		
Chertov; Oleg	Frederick	MD		
Taub; Dennis D.	Thurmont	MD		
Xu; Luoling	London			CA
Wang; Ji Ming	Frederick	MD		
Murphy; William J.	Frederick	MD		

US-CL-CURRENT: 424/185.1; 424/198.1, 514/12, 530/324

Hit List

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Search Results - Record(s) 1 through 3 of 3 returned.

☐ 1. Document ID: US D481926 S

L9: Entry 1 of 3 File: USPT Nov 11, 2003

US-PAT-NO: D481926
DOCUMENT-IDENTIFIER: US D481926 S

TITLE: Plate with adjustable mounts for telephones, tools, appliances and the like

DATE-ISSUED: November 11, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Engelmayer; Juda S.	New York	NY	10002	

US-CL-CURRENT: D08/354

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 6018313 A

L9: Entry 2 of 3 File: USPT Jan 25, 2000

US-PAT-NO: 6018313
DOCUMENT-IDENTIFIER: US 6018313 A
** See image for Certificate of Correction **

TITLE: System for determining the location of mobile objects

DATE-ISSUED: January 25, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Engelmayer; Wolfgang	Bad Honnef			DE
Lindstrot; Walter	Wachtberg			DE
Raven; Paul	Graftschaft			DE
Sandmann; Stefan	Bonn			DE
Schoemakers; Guenter	Troisdorf			DE

US-CL-CURRENT: 342/357.02; 701/215

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw Desc | Ima

☐ 3. Document ID: US 4785970 A

L9: Entry 3 of 3 File: USPT Nov 22, 1988

US-PAT-NO: 4785970
DOCUMENT-IDENTIFIER: US 4785970 A

TITLE: Tissue pack

DATE-ISSUED: November 22, 1988

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Engelmayer; Gerhard	Baden			AT

US-CL-CURRENT: 221/47; 221/63

Terms	Documents
engelmayer.in.	3

Connecting via Winsock to STN

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NEWS	9	DEC 17	ELCOM reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	10	DEC 17	COMPUAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	11	DEC 17	SOLIDSTATE reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	12	DEC 17	CERAB reloaded; updating to resume; current-awareness alerts (SDIs) affected
NEWS	13	DEC 17	THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
NEWS	14	DEC 30	EPFULL: New patent full text database to be available on STN
NEWS	15	DEC 30	CAPLUS - PATENT COVERAGE EXPANDED
NEWS	16	JAN 03	No connect-hour charges in EPFULL during January and February 2005
NEWS	17	FEB 25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	18	FEB 10	STN Patent Forums to be held in March 2005
NEWS	19	FEB 16	STN User Update to be held in conjunction with the 229th ACS National Meeting on March 13, 2005
NEWS	20	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	21	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	22	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	23	MAR 02	GBFULL: New full-text patent database on STN
NEWS	24	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	25	MAR 03	MEDLINE file segment of TOXCENTER reloaded

NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

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FILE 'HOME' ENTERED AT 15:53:22 ON 16 MAR 2005

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=> s lactoferrin and (reduce circulating levels of cholesterol?)
9 FILES SEARCHED...
L1 1 LACTOFERRIN AND (REDUCE CIRCULATING LEVELS OF CHOLESTEROL?)

=> d l1 ti abs ibib tot

L1 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2005 ACS on STN
TI **Lactoferrin** in the reduction of circulating cholesterol, vascular inflammation, atherosclerosis and cardiovascular disease
AB The invention discloses methods for using **lactoferrin** to **reduce circulating levels** of **cholesterol** and vascular inflammation in order to treat, prevent or reduce the incidence of atherosclerosis and cardiovascular disease.
ACCESSION NUMBER: 2004:490705 HCAPLUS
DOCUMENT NUMBER: 141:33800
TITLE: **Lactoferrin** in the reduction of circulating

cholesterol, vascular inflammation, atherosclerosis and cardiovascular disease
INVENTOR(S): Varadhachary, Atul; Glynn, Peter; Wang, Yenyun; Engelmayer, Jose
PATENT ASSIGNEE(S): Agennix Incorporated, USA
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004050037	A2	20040617	WO 2003-US38540	20031204
WO 2004050037	A3	20040812		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004152623	A1	20040805	US 2003-728275	20031204
PRIORITY APPLN. INFO.:			US 2002-430867P P	20021204
			US 2003-498337P P	20030827

=> s lactoferrin composition adj2 administration
L2 0 LACTOFERRIN COMPOSITION ADJ2 ADMINISTRATION

=> s lactoferrin composition
L3 18 LACTOFERRIN COMPOSITION

=> d l3 ti abs ibib tot

L3 ANSWER 1 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treating cancer, such as melanoma, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma and leukemia, by administering a cancer immunotherapy and a **lactoferrin composition**.
AN 2005-111859 [12] WPIDS
AB US2005019342 A UPAB: 20050218
NOVELTY - Treating cancer comprising administering a cancer immunotherapy and an adjuvant that is a **lactoferrin composition** administered to provide an improvement in the cancer in the subject, is new.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a method of enhancing the immune system in a subject suffering from cancer or susceptible to cancer, comprising administering to the subject a cancer immunotherapy and an adjuvant that is a **lactoferrin composition**.
ACTIVITY - Cytostatic.
BALB/Cys mice were challenged subcutaneously in the middle of the left flank with 0.2 ml of a single-cell suspension containing 1x105 Her-2/neu+Transplantable carcinoma (TUBO) cells. Oral lactoferrin or placebo was administered two days before TUBO injection and for 3 weeks. Tumors were measured twice a week for the duration of the experiment. The results showed that mice treated with oral LF displayed a significant tumor inhibition, whereas no activity was observed in mice treated with placebo or left untreated.
MECHANISM OF ACTION - Vaccine.
USE - The methods and compositions of the present invention are useful for diagnosing, preventing, staging and/or treating cancer and tumor disorders, including melanoma, non-small cell lung, small cell lung, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma, leukemia,

neuroblastoma, squamous cell, head, neck, gum, tongue, breast, pancreatic, prostate, renal, bone, testicular, ovarian, mesothelioma, sarcoma, cervical, gastrointestinal, lymphoma, brain, colon and bladder, preferably hematopoietic neoplasm such as acute myelogenous leukemia, acute lymphoblastic leukemia, myelodysplastic syndrome, chronic myelomonocytic leukemia, juvenile myelomonocyte leukemia, multiple myeloma and chronic lymphocytic leukemia.

Dwg.0/4

ACCESSION NUMBER: 2005-111859 [12] WPIDS
DOC. NO. CPI: C2005-037401
TITLE: Treating cancer, such as melanoma, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma and leukemia, by administering a cancer immunotherapy and a **lactoferrin composition**.
DERWENT CLASS: B04 D16
INVENTOR(S): PERICLE, F; VARADHACHARY, A
PATENT ASSIGNEE(S): (AGEN-N) AGENNIX INC
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2005019342	A1	20050127	(200512)*		22

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2005019342	A1 Provisional	US 2003-476318P	20030606
	Provisional	US 2003-498236P	20030827
		US 2004-862213	20040607

PRIORITY APPLN. INFO: US 2004-862213 20040607; US 2003-476318P 20030606; US 2003-498236P 20030827

L3 ANSWER 2 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Thermally stable **lactoferrin composition** for use in food/beverage products, feeds and pharmaceuticals, comprises lactoferrin blended with a stabilizer.
AN 2005-025090 [03] WPIDS
AB JP2004352669 A UPAB: 20050112
NOVELTY - A thermally stable **lactoferrin composition** contains lactoferrin blended with a stabilizer.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for food/beverage product, feed and pharmaceutical, which contain the new composition.

ACTIVITY - Antiinflammatory; Immunostimulant. No biological data is given.

MECHANISM OF ACTION - Antioxidant.

USE - In food/beverage products, feeds and pharmaceuticals (claimed).

ADVANTAGE - The **lactoferrin composition** has excellent thermal stability at 90 deg. C or more and is effectively utilized in food/beverage product, feed and pharmaceutical.

Dwg.0/0

ACCESSION NUMBER: 2005-025090 [03] WPIDS
DOC. NO. CPI: C2005-008585
TITLE: Thermally stable **lactoferrin composition** for use in food/beverage products, feeds and pharmaceuticals, comprises lactoferrin blended with a stabilizer.
DERWENT CLASS: A96 B04 D13
PATENT ASSIGNEE(S): (SNOW) SNOW BRAND MILK PROD CO LTD
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

JP 2004352669	A	20041216 (200503)*	18

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
JP 2004352669	A	JP 2003-153317	20030529

PRIORITY APPLN. INFO: JP 2003-153317 20030529

L3 ANSWER 3 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Use of a **lactoferrin composition** in the treatment of diabetes mellitus and for modulating symptoms of diabetes mellitus e.g. obesity, hyperglycemia and high blood pressure.
AN 2004-834158 [82] WPIDS
AB WO2004103285 A UPAB: 20041223
NOVELTY - Modulating and treating diabetes mellitus, and reducing blood glucose in a subject suffering from diabetes mellitus, comprises administering a **lactoferrin composition**.

ACTIVITY - Antidiabetic; Anorectic; Hypotensive. Recombinant Human Lactoferrin (RhLF) (650 mg/kg) (A) and placebo (rhLF diluent buffer pH 7.0) (B) were administered orally once daily for 15 consecutive days to groups of 4 non-insulin dependent diabetic mellitus (NIDDM) male mice. The NIDDM mice had serum glucose of -560 mg/dl. All animals were allowed for free access to normal laboratory chow and water. Blood samples were withdrawn from the orbital sinus immediately before dosing on day 1 and 90 minutes after the first administration on day 15. The animals were fasted for 3 hours prior to sampling. Four animals per cohort were studied. After 15 days, the serum glucose levels for (A)/control was found to be about 460/above 575 mg/dl. After 15 days, oral administration of (A) resulted in a 19.0% reduction in serum glucose as compared to the control composition.

MECHANISM OF ACTION - None given.

USE - The method is used:

(i) for modulation of symptoms of diabetes mellitus;

(ii) in the treatment of diabetes mellitus e.g. non-insulin dependent diabetes mellitus and insulin dependent diabetes mellitus;

(iii) for reducing blood glucose in a subject suffering from diabetes mellitus; and

(iv) for modulating blood insulin in a subject suffering from diabetes mellitus.

The symptoms include obesity, hyperglycemia and increased or decreased insulin levels and high blood pressure (claimed).

ADVANTAGE - The compound:

(i) modulates the level of insulin and blood glucose;

(ii) increases the level of insulin;

(iii) decreases the level of blood glucose; and

(iv) reduces blood pressure and total body weight.

The composition modulates blood insulin and effectively treats diabetes mellitus.

Dwg.0/4

ACCESSION NUMBER: 2004-834158 [82] WPIDS
DOC. NO. CPI: C2004-289685
TITLE: Use of a **lactoferrin composition** in the treatment of diabetes mellitus and for modulating symptoms of diabetes mellitus e.g. obesity, hyperglycemia and high blood pressure.
DERWENT CLASS: B04
INVENTOR(S): ENGELMAYER, J; VARADHACHARY, A
PATENT ASSIGNEE(S): (ENGE-I) ENGELMAYER J; (VARA-I) VARADHACHARY A; (AGEN-N) AGENNIX INC
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004103285	A2	20041202 (200482)*		EN	32

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE
LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE
DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG
KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ
OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG
US UZ VC VN YU ZA ZM ZW
US 2005004006 A1 20050106 (200504)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004103285	A2	WO 2004-US14985	20040513
US 2005004006	A1 Provisional	US 2003-470549P	20030514
		US 2004-844865	20040513

PRIORITY APPLN. INFO: US 2003-470549P 20030514; US 2004-844865 20040513

L3 ANSWER 4 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treating subject suffering from pain, involves administering **lactoferrin composition**, to provide improvement in pain in subjects.
AN 2004-488007 [46] WPIDS
AB WO2004054608 A UPAB: 20040720
NOVELTY - Treating (M1) a subject suffering from pain, involves administering to the subject a **lactoferrin composition** , to provide an improvement in pain in the subject.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for modulating acute pain or chronic pain in a subject, involves administering **lactoferrin composition** to the subject to provide an improvement in acute pain or chronic pain in the subject.
ACTIVITY - Analgesic.
The ability of recombinant human lactoferrin (rhLF) to reduce heat-induced pain in tail flick test in mice was done as follows. Mice received 1000 mg/kg rhLF or placebo orally 60 minutes prior to test (N = 5 per group). The tail flick test measured the time (max 15 seconds) required to elicit the radiation heat-induced tail-flick response in mice. Result showed that oral lactoferrin treatment significantly reduced pain in mice.
MECHANISM OF ACTION - Reduces production or activity of pro-inflammatory cytokines; Enhances production or activity of cytokines (claimed).
USE - (M1) is useful for treating a subject suffering from pain (acute or chronic pain) (claimed).
DESCRIPTION OF DRAWING(S) - The figure is a graph showing reduction in heat-induced pain measured by tail flick test in mice.
Dwg.1/2
ACCESSION NUMBER: 2004-488007 [46] WPIDS
DOC. NO. CPI: C2004-181880
TITLE: Treating subject suffering from pain, involves administering **lactoferrin composition** , to provide improvement in pain in subjects.
DERWENT CLASS: B04 D16
INVENTOR(S): PETRAK, K; VARADHACHARY, A
PATENT ASSIGNEE(S): (PETR-I) PETRAK K; (VARA-I) VARADHACHARY A; (AGEN-N) AGENNIX INC
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004054608	A2	20040701	(200446)*	EN	30

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE
LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE

DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG
KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM
PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US
UZ VC VN YU ZA ZM ZW
US 2004151784 A1 20040805 (200452)
AU 2003293500 A1 20040709 (200474)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004054608	A2	WO 2003-US39358	20031211
US 2004151784	A1 Provisional	US 2002-432937P	20021212
	Provisional	US 2003-498248P	20030827
		US 2003-733621	20031211
AU 2003293500	A1	AU 2003-293500	20031211

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003293500	A1 Based on	WO 2004054608

PRIORITY APPLN. INFO: US 2003-498248P 20030827; US
2002-432937P 20021212; US
2003-733621 20031211

L3 ANSWER 5 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treating tissue or organ (e.g., kidney, heart, liver, lung or pancreas) transplant rejection in recipient involves administering a **lactoferrin composition** to the recipient to attenuate tissue or organ transplant rejection.
AN 2004-468695 [44] WPIDS
AB WO2004052305 A UPAB: 20040712
NOVELTY - Treating a tissue or organ transplant rejection in a recipient involves administering to the recipient, a **lactoferrin composition** to attenuate the tissue or organ transplant rejection.
ACTIVITY - Immunosuppressive.
MECHANISM OF ACTION - Reducer of allogenic immune responses in recipient; Regulator of T cell responses; Stimulator of interleukin-18 or MIP-3- alpha in the gastrointestinal tract; Regulator of activity of B and T lymphocytes, antigen-presenting cells, natural killer cells, macrophages and granulocytes; Regulator of production or activity of pro-inflammatory cytokines (all claimed). Heterotopic heart transplantation in 8-10 weeks old rats (BUF, donor to WF, recipient) was performed using standard microsurgical technique of end-to-side anastomoses to recipient aorta and vena cava. Graft survival was defined as the last day of transabdominally palpable cardiac contractions. Recipients were treated with either placebo or recombinant human lactoferrin (rhLF) (625 mg/Kg) for 14 days starting seven days prior to the transplant. The results showed that lactoferrin alone significantly extended cardiac allograft survival.
USE - For treating tissue (bone marrow or peripheral stem cells) or organ (kidney, heart, lung, liver, or pancreas) transplant rejection in the recipient (claimed).
ADVANTAGE - The method induces permanent allograft or xenograft acceptance and reducing the incidence of graft-versus-host-disease involved in bone marrow or peripheral stem cells transplantation.
Dwg.0/3
ACCESSION NUMBER: 2004-468695 [44] WPIDS
DOC. NO. CPI: C2004-175661
TITLE: Treating tissue or organ (e.g., kidney, heart, liver, lung or pancreas) transplant rejection in recipient involves administering a **lactoferrin composition** to the recipient to attenuate tissue or organ transplant rejection.
DERWENT CLASS: B04 D16
INVENTOR(S): PERICLE, F; VARADHACHARY, A
PATENT ASSIGNEE(S): (PERI-I) PERICLE F; (VARA-I) VARADHACHARY A; (AGEN-N)

AGENNIX INC
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004052305	A2	20040624	(200444)*	EN	38
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2004176276	A1	20040909	(200459)		
AU 2003296447	A1	20040630	(200472)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004052305	A2	WO 2003-US39265	20031210
US 2004176276	A1 Provisional	US 2002-432113P	20021210
	Provisional	US 2003-498338P	20030827
		US 2003-732429	20031210
AU 2003296447	A1	AU 2003-296447	20031210

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003296447	A1 Based on	WO 2004052305

PRIORITY APPLN. INFO: US 2003-498338P 20030827; US 2002-432113P 20021210; US 2003-732429 20031210

L3 ANSWER 6 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treating bacteremia, involves administering **lactoferrin composition** orally to subject to provide improvement in bacteremia of subject.
AN 2004-468687 [44] WPIDS
AB WO2004052281 A UPAB: 20040712
NOVELTY - Treating (M1) bacteremia, involves administering **lactoferrin composition** orally to a subject to provide an improvement in the bacteremia of the subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) treating bacteremia or sepsis, involves supplementing the mucosal immune system in a subject by administering **lactoferrin composition** through an oral route;

(2) enhancing (M2) a mucosal immune response in the gastrointestinal tract in a subject, involves administering **lactoferrin composition** orally to the subject;

(3) decreasing mortality of a subject having bacteremia, involves administering **lactoferrin composition** orally to the subject to attenuate the bacteremia to decrease mortality of the subject;

(4) treating (M3) a septic condition in a subject, involves administering **lactoferrin composition** orally to the subject to provide an improvement in the septic condition of the subject;

(5) decreasing (M4) mortality of a subject having sepsis, involves administering **lactoferrin composition** orally to the subject to attenuate sepsis to decrease mortality of the subject; and

(6) decreasing (M5) mortality of a subject having acute lung injury (ALI) or acute respiratory distress syndrome (ARDS), involves administering **lactoferrin composition** orally to the subject to attenuate ALI or ARDS to decrease mortality of the subject.

ACTIVITY - Antibacterial; Immunosuppressive; Respiratory-Gen.; Antiinflammatory.

In vivo analysis of recombinant lactoferrin in murine lipopolysaccharide (LPS) model of sepsis, was carried out as follows. Groups of mice were used for the study. Animals received different doses of Escherichia coli LPS (30, 20, 15 and 10 ng/mouse) and vehicle (saline, 0.2 ml/mouse) immediately after pre-treatment with D(+)- galactosamine (20 mg/mouse). Mortality was recorded every 12 hours over a 3-day period. The LPS (20-30 ng/mouse) resulted in 100% mortality and 15 ng/mouse resulted in 50% mortality. Vehicle and test substance comprising recombinant human lactoferrin (rhLF) were administered intravenously to groups of 8 male mice weighing 18-20 g, 16 minutes before and 10 minutes after challenge with LPS plus galactosamine. The result indicated a reduction in mortality induced by LPS by 38%.

MECHANISM OF ACTION - Enhancer of mucosal immune response; Stimulator of IL-18; Stimulator of immune cell production; Reduces production or activity of inflammatory cytokines (claimed).

USE - (M1) is useful for treating bacteremia in a subject, where the improvement includes attenuating sepsis, septic shock and organ failure. The **lactoferrin composition** of (M1) is useful for treating sepsis and for decreasing mortality a subject having ALI or ARDS (claimed).

The **lactoferrin composition** of (M1) is useful for treating a subject suspected of or having bacteremia, sepsis or septic shock caused by Gram-negative bacteria such as Escherichia, Shigella and Salmonella, Gram-positive bacteria such as Staphylococcus aureus and Bacillus sp. or other infectious agents. The **lactoferrin composition** of (M1) is useful for treating or preventing the consequences of bacterially induced systemic inflammatory response syndrome, and for treating or preventing endotoxemia.

ADVANTAGE - (M1) enables improvement in the bacteremia of the subject, where the improvement includes attenuating sepsis, septic shock or organ failure (claimed), decreasing days of hospitalization, decreasing or eliminating intensive care such as intensive care unit, or decreasing or eliminating the use of supportive care such as a mechanical ventilator. The **lactoferrin composition** of (M1) is easily administered in a variety of dosage forms to result in an improvement or remediation of the symptoms.

DESCRIPTION OF DRAWING(S) - The figure is a graph showing the reduction of mortality and key cytokines in sepsis.

Dwg.2/2

ACCESSION NUMBER: 2004-468687 [44] WPIDS
DOC. NO. CPI: C2004-175653
TITLE: Treating bacteremia, involves administering **lactoferrin composition** orally to subject to provide improvement in bacteremia of subject.
DERWENT CLASS: B04
INVENTOR(S): PETRAK, K; VARADHACHARY, A
PATENT ASSIGNEE(S): (PETR-I) PETRAK K; (VARA-I) VARADHACHARY A; (AGEN-N) AGENNIX INC
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004052281	A2	20040624	(200444)*	EN	44
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2004152624	A1	20040805	(200452)		
AU 2003298906	A1	20040630	(200472)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 2004052281	A2		WO 2003-US38621	20031205
US 2004152624	A1	Provisional	US 2002-431393P	20021206
		Provisional	US 2003-498327P	20030827
			US 2003-728521	20031205
AU 2003298906	A1		AU 2003-298906	20031205

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003298906	A1 Based on	WO 2004052281

PRIORITY APPLN. INFO: US 2003-498327P 20030827; US 2002-431393P 20021206; US 2003-728521 20031205

L3 ANSWER 7 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Treating a cardiovascular disease comprises administering to a subject an effective amount of a **lactoferrin composition** to provide an improvement in the cardiovascular disease in the subject.

AN 2004-460986 [43] WPIDS

AB WO2004050037 A UPAB: 20040709

NOVELTY - Treating a cardiovascular disease comprises administering to a subject an effective amount of a **lactoferrin composition** to provide an improvement in the cardiovascular disease in the subject.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method of modulating atherosclerosis in a subject comprising administering to the subject an effective amount of a **lactoferrin composition** to modulate atherosclerosis in the subject.

ACTIVITY - Cardiant; Antiarteriosclerotic. No biological data given.

MECHANISM OF ACTION - Gene therapy; HMG-coA reductase inhibitor.

USE - The method is useful for treating a cardiovascular disease, e.g. atherosclerosis (claimed).

Dwg.0/5

ACCESSION NUMBER: 2004-460986 [43] WPIDS

DOC. NO. CPI: C2004-172138

TITLE: Treating a cardiovascular disease comprises administering to a subject an effective amount of a **lactoferrin composition** to provide an improvement in the cardiovascular disease in the subject.

DERWENT CLASS: B04 D16

INVENTOR(S): ENGELMAYER, J; GLYNN, P; VARADHACHARY, A; WANG, Y

PATENT ASSIGNEE(S): (ENGE-I) ENGELMAYER J; (GLYN-I) GLYNN P; (VARA-I) VARADHACHARY A; (WANG-I) WANG Y; (AGEN-N) AGENNIX INC

COUNTRY COUNT: 107

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004050037	A2	20040617	(200443)*	EN	38
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2004152623	A1	20040805	(200452)		
AU 2003291206	A1	20040623	(200472)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2004050037	A2		WO 2003-US38540	20031204
US 2004152623	A1	Provisional	US 2002-430867P	20021204
		Provisional	US 2003-498337P	20030827
			US 2003-728275	20031204

AU 2003291206 A1 AU 2003-291206 20031204

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003291206	A1 Based on	WO 2004050037

PRIORITY APPLN. INFO: US 2003-498337P 20030827; US 2002-430867P 20021204; US 2003-728275 20031204

L3 ANSWER 8 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Treatment of hyperproliferative disease, e.g. cancer, rheumatoid arthritis, inflammatory bowel disease, or leiomyomas, involves supplementing systemic or local immune system by increasing lactoferrin.

AN 2004-071004 [07] WPIDS

CR 2004-035048 [03]

AB WO2003094952 A UPAB: 20040702

NOVELTY - Treatment of hyperproliferative disease involves supplementing a systemic or local immune system by increasing a lactoferrin at the site of the hyperproliferative disease.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(a) enhancing a local immune response in the vicinity of a tumor following the step of administering intratumorally the **lactoferrin composition** (C1); and

(b) treatment of hyperproliferative disease involving administering (C1) in combination with chemotherapy, biotherapy, immunotherapy, surgery or radiotherapy.

ACTIVITY - Cytostatic; Antiarthritic; Antirheumatic; Antiinflammatory; Osteopathic; Gastrointestinal-Gen.; Vasotropic; Antiarteriosclerotic; Antipsoriatic; Immunomodulator; Dermatological; Immunosuppressive.

MECHANISM OF ACTION - Tumor cell growth inhibitor; Interleukin-18 or GM-CSF production stimulator. The tumor cell growth inhibitory efficacy of recombinant human lactoferrin (rhLF) was tested on human squamous cell carcinoma (O12). The cells were injected into the right flank of athymic nude mice and rhLF (test) was administered orally at a dosage of 20 mg/dose twice a day for five days starting 11 days after inoculation with tumor cells. Control animals were treated with only the vehicle. The efficacy of the treatment was evaluated by measuring the solid tumor size during and at the end of the test. The result showed that the oral treatment with rhLF significantly reduced the tumor growth by 80% compared to the control.

USE - For treating hyperproliferative disease, e.g. cancer including neoplasm (e.g. melanoma, non-small cell lung, small cell lung, lung hepatocarcinoma, retinoblastoma, astrocytoma, gliobastoma, leukemia, neuroblastoma, squamous cell, head, neck, gum, tongue, breast, pancreatic, prostate, renal, bone, testicular, ovarian, mesothelioma, sarcoma, cervical, gastrointestinal, lymphoma, brain, colon and bladder); rheumatoid arthritis; inflammatory bowel disease; osteoarthritis; leiomyomas; adenomas; lipomas; hemangiomas; fibromas; vascular occlusion; restenosis; atherosclerosis; pre-neoplastic lesions; carcinoma in situ; oral hairy leukoplakia and psoriasis (claimed). Also useful for treating neurofibromatosis, Waginer's granulomatosis, Kawasaki's disease, lupus erythematosus and midline granuloma.

ADVANTAGE - The lactoferrin stimulates the production of interleukin-18 or GM-CSF in the site of injection, which stimulates the production, maturation or activity of immune cells (e.g. T lymphocytes selected from CD4+, CD8+ or CD3+ cells), dendritic or other antigen presenting cells.

Dwg.0/5

ACCESSION NUMBER: 2004-071004 [07] WPIDS

CROSS REFERENCE: 2004-035048 [03]

DOC. NO. CPI: C2004-029321

TITLE: Treatment of hyperproliferative disease, e.g. cancer, rheumatoid arthritis, inflammatory bowel disease, or

leiomyomas, involves supplementing systemic or local immune system by increasing lactoferrin.

DERWENT CLASS: B04
INVENTOR(S): BARSKY, R; O'MALLEY, B; PETRAK, K; VARADHACHARY, A
PATENT ASSIGNEE(S): (AGEN-N) AGENNIX INC
COUNTRY COUNT: 103
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003094952	A1	20031120	(200407)*	EN	23

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

AU 2003239393	A1	20031111	(200442)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003094952	A1	WO 2003-US14584	20030509
AU 2003239393	A1	AU 2003-239393	20030509

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2003239393	A1	Based on	WO 2003094952

PRIORITY APPLN. INFO: US 2002-379474P 20020510; US 2002-379441P 20020510; US 2002-379442P 20020510

L3 ANSWER 9 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Use of a **lactoferrin composition** for the treatment of a respiratory disorder e.g. asthma, emphysema, bronchitis, chronic obstructive pulmonary disease.

AN 2004-042695 [04] WPIDS

AB WO20,03099207 A UPAB: 20040115

NOVELTY - Treatment (M1) of a respiratory disorder comprising administrating a **lactoferrin composition** (c1), is new.

ACTIVITY - Antiasthmatic; Antiinflammatory; Antiallergic; Respiratory-Gen.

Allergic sheep were treated with recombinant human lactoferrin (rhLF) and compared to their historic controls. Baseline bronchoalveolar lavage fluid (BAL) samples were taken from all animals and baseline dose response curves to aerosol carbachol were obtained in all sheep 1 - 3 days before the start of dosing. The sheep were pre-treated with oral rhLF (1 or 1.5 g) twice daily for 3 days prior to allergen challenge. Plasma samples were taken at both the time of 1st dose on day 1 and again on day 3 at the time of the second daily dose. On the challenge day (day 4) the test sheep received rhLF in dose of 1 g or 1.5 g 30 minutes before, 4 hours after and 24 hours after allergen challenge. BAL samples were again taken and additional plasma samples were also taken before and immediately after rhLF administration. On the challenge day (day 4) measurements of lung resistance (RL) were obtained before and then repeated 30 minutes after treatment and then the sheep were challenged with Ascaris suum allergen. Measurements of RL were obtained immediately after challenge, hourly form 1 - 8 hours after challenge. The RL (%) (for rhLF(1 g)/rhLF(1.5 g)/control) was 350/550/500 (at 0 hour), 0/0/0 (after 4 hours) and 0/20/100 (after 8 hours). The peak delayed increase in RL (%) was 76/74/0; the delayed airway hypersensitivity (%) was 100/88/0; the % increase in total inflammatory cells in the lungs was -/100/0 for rhLF(1 g)/rhLF(1.5 g)/control respectively.

MECHANISM OF ACTION - Interleukin-18 stimulator; Stimulator of production or activity of immune cells (preferably T lymphocytes and natural killer cells, especially CD4+, CD8+ and CD3+ cells); Granulocyte Macrophage Colony Stimulating Factor (GM-CSF) stimulator; Stimulator of production, maturation or activity of immune cells (preferably dendritic or other antigen representing cells); Inhibitor of production or activity of pro-inflammatory cytokines.

The effect of oral administration of recombinant human lactoferrin (rhLF) on GM-CSF was tested in vivo. Mice (5 per group) were treated for 3 days daily with 300 mg/kg/day of rhLF. For a control, mice were only administered a pharmaceutical carrier. Twenty-four hours after administration of the LF or placebo for 3 days, animals were sacrificed and the small intestine tissue was removed for further analysis. Small intestinal epithelium was homogenized using a lysis buffer consisting of phosphate buffered saline (PBS), 1% Nonidet P-40, 0.5% sodium deoxycholate and 0.1% sodium dodecyl sulfate containing phenylmethylsulfonyl fluoride (10 micro g/ml). Homogenate was centrifuged for 10 minutes and was tested for GM-CSF levels. The rhLF increased the production of a key immunostimulatory cytokine, GM-CSF in the small intestine compared to placebo (19.4%).

USE - The method is useful for the treatment of allergic or non-allergic respiratory disorder including atopic asthma, non-atopic asthma, emphysema, bronchitis, chronic obstructive pulmonary disease, acute or chronic sinusitis, and allergic rhinitis (all claimed).

ADVANTAGE - (c1) enhances a mucosal immune response in the gastrointestinal tract; reduces the infiltration of inflammatory cells into the lung; reduces the delayed hypersensitivity associated with atopic or non-atopic asthma. The lactoferrin stimulates interleukin-18 in the gastrointestinal tract; stimulates the production, maturation or activity of immune cells (e.g. T lymphocytes (e.g. CD4+, CD8+, or CD3+ cells) or natural killer cells; stimulates GM-SCF in the gastrointestinal tract, thus stimulating the production, maturation or activity of immune cells (e.g. dendritic or other antigen presenting cells) and reduces the production or activity of pro-inflammatory cytokines.

Dwg.0/12

ACCESSION NUMBER: 2004-042695 [04] WPIDS
DOC. NO. CPI: C2004-017566
TITLE: Use of a **lactoferrin composition** for the treatment of a respiratory disorder e.g. asthma, emphysema, bronchitis, chronic obstructive pulmonary disease.
DERWENT CLASS: B05 B07 C03 C07 D16
INVENTOR(S): GLYNN, P; VARADHACHARY, A
PATENT ASSIGNEE(S): (GLYN-I) GLYNN P; (VARA-I) VARADHACHARY A; (AGEN-N) AGENNIX INC
COUNTRY COUNT: 103
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003099207	A2	20031204	(200404)*	EN	28
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2004009896	A1	20040115	(200406)		
AU 2003233583	A1	20031212	(200443)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003099207	A2	WO 2003-US15763	20030520
US 2004009896	A1 Provisional	US 2002-383280P	20020524
	Provisional	US 2002-410645P	20020913

		US 2003-441329	20030520
AU 2003233583	A1	AU 2003-233583	20030520

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003233583	A1 Based on	WO 2003099207

PRIORITY APPLN. INFO: US 2002-410645P 20020913; US 2002-383280P 20020524; US 2003-441329 20030520

L3 ANSWER 10 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Treating a hyperproliferative disease (e.g. cancer, psoriasis, adenoma or atherosclerosis) in a subject comprises administering a composition of a human lactoferrin alone or in combination with standard anti-cancer therapies.

AN 2004-035048 [03] WPIDS

CR 2004-071004 [07]

AB WO2003099323 A UPAB: 20050303

NOVELTY - Treating a hyperproliferative disease comprises administering orally, intravenously or topically to a subject a human **lactoferrin composition** in an amount sufficient to provide an improvement in the hyperproliferative disease in the subject.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a method of enhancing a mucosal immune response in the gastrointestinal tract in a subject, comprising administering orally to the subject a human lactoferrin;

(2) a method of reducing growth of a neoplasm in a subject, comprising administering orally to the subject a human **lactoferrin composition** in an amount to reduce the growth of the neoplasm in the subject;

(3) methods of enhancing a systemic or local immune response following the step of administering intravenously or topically to the subject a **lactoferrin composition**; and

(4) methods of stimulating interleukin-18 or GFM-CSF in a subject, comprising administering to the subject the **lactoferrin composition**.

ACTIVITY - Cytostatic; Antirheumatic; Antiarthritic; Antiinflammatory; Osteopathic; Vasotropic; Antiarteriosclerotic; Antipsoriatic. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The methods are useful in treating malignant neoplasms (e.g. melanoma or leukemia) and other hyperproliferative diseases such as rheumatoid arthritis, inflammatory bowel disease, osteoarthritis, leiomyomas, adenomas, lipomas, hemangiomas, fibromas, vascular occlusion, restenosis, atherosclerosis, pre-neoplastic lesions, carcinoma in situ, oral hairy leukoplakia or psoriasis.

Dwg.0/5

ACCESSION NUMBER:	2004-035048 [03] WPIDS
CROSS REFERENCE:	2004-071004 [07]
DOC. NO. CPI:	C2004-011624
TITLE:	Treating a hyperproliferative disease (e.g. cancer, psoriasis, adenoma or atherosclerosis) in a subject comprises administering a composition of a human lactoferrin alone or in combination with standard anti-cancer therapies.
DERWENT CLASS:	B04
INVENTOR(S):	BARSKY, R; PERICLE, F; PETRAK, K; VARADHACHARY, A; WANG, Y; O'MALLEY, B
PATENT ASSIGNEE(S):	(BARS-I) BARSKY R; (PERI-I) PERICLE F; (PETR-I) PETRAK K; (VARA-I) VARADHACHARY A; (WANG-I) WANG Y; (OMAL-I) O'MALLEY B; (AGEN-N) AGENNIX INC
COUNTRY COUNT:	104
PATENT INFORMATION:	

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003099323	A1	20031204	(200403)*	EN	51
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
US 2004009895	A1	20040115	(200406)		
US 2004082504	A1	20040429	(200429)		
AU 2003273182	A1	20031212	(200443)		
EP 1507554	A1	20050223	(200515)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2003099323	A1		WO 2003-US14789	20030509
US 2004009895	A1	Provisional	US 2002-379441P	20020510
		Provisional	US 2002-379442P	20020510
		Provisional	US 2002-379474P	20020510
			US 2003-434769	20030509
US 2004082504	A1	Provisional	US 2002-379441P	20020510
		Provisional	US 2002-379442P	20020510
		Provisional	US 2002-379474P	20020510
			US 2003-435319	20030509
AU 2003273182	A1		AU 2003-273182	20030509
EP 1507554	A1		EP 2003-755357	20030509
			WO 2003-US14789	20030509

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2003273182	A1	Based on	WO 2003099323
EP 1507554	A1	Based on	WO 2003099323

PRIORITY APPLN. INFO: US 2002-379474P 20020510; US 2002-379441P 20020510; US 2002-379442P 20020510; US 2003-434769 20030509; US 2003-435319 20030509

L3 ANSWER 11 OF 18 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Immobilized **lactoferrin composition** comprises natural substrate and carrier useful as antimicrobicide e.g. for preventing food poisoning or diarrhea or in cosmetics and cleaners.

AN 2001-041106 [05] WPIDS

CR 2001-061417 [07]; 2004-267982 [25]

AB WO 200072874 A UPAB: 20041006

NOVELTY - Composition comprises a defined dispersion of lactoferrin immobilized on a natural substrate via the N-terminus region of the lactoferrin and a carrier.

DETAILED DESCRIPTION - Composition comprises:

(a) a defined dispersion of lactoferrin immobilized on a natural substrate via the N-terminus region of the lactoferrin; and

(b) a carrier.

INDEPENDENT CLAIMS are also included for methods of preventing or inhibiting growth and/or adhesion of a microbe (i) on or in a human, (ii) on or in a non-human vertebrate subject or (iii) on a biological surface or in a biological fluid comprising treating (i)-(iii) with a composition comprising an isolated lactoferrin immobilized on a natural substrate via the N-terminus region of the lactoferrin.

ACTIVITY - Antimicrobial; Antibacterial; Fungicide; Protozoacide.

In assays a 1 % mixture of ImLF and nLF in 0.001 M citric acid, 0.01 M sodium bicarbonate and 0.1 M sodium chloride after 24 hours gave 100 % inhibition of Escherichia coli serotype O157:H7 in a 50 mu l bacterial suspension containing 106 cells/ml.

USE - As antimicrobicides useful as human or veterinary pharmaceuticals (e.g. for preventing food poisoning or treating diarrhea), cosmetics, cleansers (e.g. skin cleansers, sanitary wipes or shampoos), mouth washes, dentrifices, bandages, food supplements, preservatives for biological fluids (e.g. semen, blood, urine or cerebro-spinal fluid) or for preventing microbial growth and/or adhesion on biological surfaces (e.g. cell skin or eggshell surface)

Dwg.0/0

ACCESSION NUMBER: 2001-041106 [05] WPIDS
CROSS REFERENCE: 2001-061417 [07]; 2004-267982 [25]
DOC. NO. CPI: C2001-011971
TITLE: Immobilized **lactoferrin composition** comprises natural substrate and carrier useful as antimicrobicide e.g. for preventing food poisoning or diarrhea or in cosmetics and cleaners.
DERWENT CLASS: A96 B04 C03 D13 D16 D21
INVENTOR(S): NAIDU, A S
PATENT ASSIGNEE(S): (NAID-I) NAIDU A S; (NAID-I) NAIDU A
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000072874	A1	20001207	(200105)*	EN	60
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000054491	A	20001218	(200118)		
AU 2003204900	A1	20030724	(200464)#		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000072874	A1	WO 2000-US14820	20000526
AU 2000054491	A	AU 2000-54491	20000526
AU 2003204900	A1 Div ex	AU 2000-53035	20000526
		AU 2003-204900	20030624

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000054491	A Based on	WO 2000072874

PRIORITY APPLN. INFO: US 1999-322700 19990528; AU 2003-204900 20030624

L3 ANSWER 12 OF 18 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Treating cancer, such as melanoma, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma and leukemia, by administering a cancer immunotherapy and a **lactoferrin composition**;
involving vector-mediated immunomodulator cytokine gene transfer and expression in dendrite cell for therapy
AN 2005-07224 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Treating cancer comprising administering a cancer immunotherapy and an adjuvant that is a **lactoferrin composition** administered to provide an improvement in the cancer in the subject, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a

method of enhancing the immune system in a subject suffering from cancer or susceptible to cancer, comprising administering to the subject a cancer immunotherapy and an adjuvant that is a **lactoferrin composition**.

BIOTECHNOLOGY - Preferred Method: The **lactoferrin composition** in treating cancer is dispersed in a carrier. The lactoferrin is recombinant bovine or human lactoferrin, where the **lactoferrin composition** comprises an N-terminal lactoferrin variant that lacks at least the N-terminal glycine residue and has at least 1-50% of the **lactoferrin composition**. The amount of the lactoferrin that is administered is 10 mg to 100 g per day. The cancer is a neoplasm, such as melanoma, non-small cell lung, small cell lung, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma, leukemia, neuroblastoma, squamous cell, head, neck, gum, tongue, breast, pancreatic, prostate, renal, bone, testicular, ovarian, mesothelioma, sarcoma, cervical, gastrointestinal, lymphoma, brain, colon and bladder, preferably hematopoietic neoplasm including acute myelogenous leukemia, acute lymphoblastic leukemia, myelodysplastic syndrome, chronic myelomonocytic leukemia, juvenile myelomonocyte leukemia, multiple myeloma and chronic lymphocytic leukemia. The **lactoferrin composition** is administered orally, parenterally or topically. The immunotherapy comprises antigen presenting cells, administration of a tumor antigen or a nucleic acid sequence expressing a cancer antigen to the subject. The nucleic acid sequence is contained in a vector. The immunotherapy further comprises administration of a vector containing a nucleic acid sequence expressing an immunomodulatory cytokine or a protein or nucleic acid that promotes the recognition of a cancer antigen in the subject. The **lactoferrin composition** is administered ex vivo to the antigen presenting cells prior to administering the cells to the subject. The cells are allogeneic or syngeneic. The composition is administered simultaneously and/or sequentially with the immunotherapy. The method further comprises additionally administering chemotherapy, immunotherapy, surgery, biotherapy, radiotherapy or their combination. The lactoferrin in enhancing the immune system is recombinant human or bovine lactoferrin, and is administered orally, and stimulates the production of interleukin-18, GM-CSF or MIP-3 alpha that stimulate the production, maturation, migration or activity of immune cells, where the immune cells are T lymphocytes, natural killer cells, dendritic cells, antigen presenting cells or progenitor cells. The T lymphocytes are CD4+, CD8+ and CD3+ cells.

ACTIVITY - Cytostatic. BALB/Cys mice were challenged subcutaneously in the middle of the left flank with 0.2 ml of a single-cell suspension containing 1x105 Her-2/neu+Transplantable carcinoma (TUBO) cells. Oral lactoferrin or placebo was administered two days before TUBO injection and for 3 weeks. Tumors were measured twice a week for the duration of the experiment. The results showed that mice treated with oral LF displayed a significant tumor inhibition, whereas no activity was observed in mice treated with placebo or left untreated.

MECHANISM OF ACTION - Vaccine.

USE - The methods and compositions of the present invention are useful for diagnosing, preventing, staging and/or treating cancer and tumor disorders, including melanoma, non-small cell lung, small cell lung, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma, leukemia, neuroblastoma, squamous cell, head, neck, gum, tongue, breast, pancreatic, prostate, renal, bone, testicular, ovarian, mesothelioma, sarcoma, cervical, gastrointestinal, lymphoma, brain, colon and bladder, preferably hematopoietic neoplasm such as acute myelogenous leukemia, acute lymphoblastic leukemia, myelodysplastic syndrome, chronic myelomonocytic leukemia, juvenile myelomonocyte leukemia, multiple myeloma and chronic lymphocytic leukemia.

ADMINISTRATION - The dosage of the **lactoferrin composition** ranges from 1 mg o 100 g per day. Routes of administration of the composition include oral, subcutaneous, intramuscular, intraperitoneal, intravenous, intraarterial, transendocardial, transepicardial, intramyocardial, intrathecal and topical. (22 pages)

ACCESSION NUMBER: 2005-07224 BIOTECHDS

TITLE: Treating cancer, such as melanoma, lung hepatocarcinoma, retinoblastoma, astrocytoma, glioblastoma and leukemia, by administering a cancer immunotherapy and a **lactoferrin composition**;
involving vector-mediated immunomodulator cytokine gene transfer and expression in dendrite cell for therapy

AUTHOR: VARADHACHARY A; PERICLE F

PATENT ASSIGNEE: AGENNIX INC

PATENT INFO: US 2005019342 27 Jan 2005

APPLICATION INFO: US 2004-862213 7 Jun 2004

PRIORITY INFO: US 2004-862213 7 Jun 2004; US 2003-476318 6 Jun 2003

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2005-111859 [12]

L3 ANSWER 13 OF 18 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN

TI New beta lactoglobulin, alpha lactalbumin or cow lactoferrin derived peptide, useful for treating systemic inflammatory response syndrome, retinopathy and rheumatoid arthritis;
beta-lactoglobulin, alpha-lactalbumin or cattle **lactoferrin composition** for disease therapy

AN 2004-23477 BIOTECHDS

AB DERWENT ABSTRACT:

NOVELTY - A beta-lactoglobulin, alpha-lactalbumin or cow lactoferrin derived peptide (I) and its salt, is new.

DETAILED DESCRIPTION - A beta-lactoglobulin, alpha-lactalbumin or cow lactoferrin derived peptide (I) is chosen from (a) peptides of formula (S1)-(S4), given below; and (b) peptides of sequence Leu-Asp-Gln-Trp-Leu-Cys-Glu-Lys, Phe-Lys-Ile-Asp-Ala-Leu-Asn-Glu, Ile-Asp-Ala-Leu-Asn-Glu-Asn-Lys, Ile-Pro-Ala-Val-Phe-Lys, Ile-Pro-Ala-Val-Phe-Lys-Ile-Asp-Ala-Leu-Asn-Glu-Asn-Lys, Ile-Pro-Ala-Val-Phe-Lys-Ile-Asp-Ala-Leu-Asn-Glu, Glu-Thr-Ala-Glu-Glu-Val-Lys, Leu-Gly-Ala-Pro-Ser-Ile-Thr-Cys-Val-Arg, Trp-Gln-Trp-Arg, and Glu-Asp-Leu-Ile-Trp-Lys. X1-Leu-Ala-His-Lys-X2-X3 (S1), where, X1 = absent or Trp; X2 = absent or Ala; X3 = absent or Leu. Y1-Leu-Pro-Met-His-Y2-Y3, (S2) where, Y1 = absent or Ala; Y2 = absent or Ile; Y3 = absent or Arg. Y4-Ile-Asp-Ala-Leu-Asn-Glu-Y5, (S3) where, Y4 = absent or Lys; Y5 = absent or Asn. Ile-Pro-Ala-Val-Phe-Lys-Y6-Y7-Y8-Y9-Y10, (S4) where, Y6 = absent or Ile; Y7 = absent or Asp; Y8 = absent or Ala; Y9 = absent or Leu; Y10 = absent or Asn. INDEPENDENT CLAIMS are also included for: (1) an (anti-inflammatory) pharmaceutical containing (I); and (2) an oral or enteral nutritive composition (C1) for treating inflammatory reactions including systemic inflammatory response syndrome which accompanies biological invasion, comprising (I) or its salt.

ACTIVITY - Antiarthritic; Antiinflammatory; Antimicrobial; Antirheumatic; Antiulcer; Cardiant; Dermatological; Gastrointestinal-Gen.; Hepatotropic; Immunosuppressive; Nephrotropic; Neuroprotective; Ophthalmological; Virucide; Vulnerary.

MECHANISM OF ACTION - TNF synthesis inhibitor; IL-6 synthesis inhibitor. In vivo analysis of inhibition of IL-6 production by peptides derived from beta-lactoglobulin and alpha-lactalbumin was carried out as follows. The 6-week old male mice were taken. They were divided into beta-lactoglobulin trypsin digested product administration group (5 mg/mouse) and alpha-lactalbumin trypsin digested product administration group (5 mg/mouse). The peptide comprising a sequence of Phe-Lys-Ile-Asp-Ala-Leu-Asn-Glu derived from beta-lactoglobulin and a peptide having a sequence of Leu-Asp-Gln-Trp-Leu-Cys-Glu-Lys derived from alpha-lactalbumin, were orally administered into the mice. The lipopolysaccharide was intraperitoneally administered at a concentration of 50 microgram/mouse. After 90 minutes, blood was obtained from the mice and blood serum was collected by centrifugation. The IL-6 level in the blood serum was measured by enzyme linked immunosorbent assay (ELISA). The result indicated reduction in the concentration of IL-6 level in the beta-lactoglobulin trypsin digested product administered group and alpha-lactalbumin trypsin digested product administered group.

USE - Peptides (I) are useful for manufacturing a pharmaceutical. (I) and their salts are useful for manufacturing an anti-inflammatory agent, and for manufacturing oral and enteral nutritive composition for

treating inflammatory reaction including systemic inflammatory response syndrome, which accompanies biological invasion such as surgery, external injury, thermal burn, infectious disease, acute pancreatitis, liver failure, peritonitis or malignant tumor (claimed). (I) is useful in treating or preventing inflammatory diseases such as retinopathy, nephropathy, neuropathy, rheumatoid arthritis, myelitis, inflammatory bowel disease, Crohn's disease, ulcerative colitis, systemic lupus erythematosus, myocardial infarction, diseases caused by Herpesvirus and influenza virus, etc.

ADMINISTRATION - A pharmaceutical comprising (I), is administered by oral route (claimed). No specific dosage details are given.

ADVANTAGE - (I) has an inflammatory cytokine production inhibitory activity. (I) is effective in the treatment or prevention of inflammatory diseases resulting from an abnormal production of TNF-alpha and interleukin (IL)-6.

EXAMPLE - Whey protein isolated substance (containing beta-lactoglobulin and alpha-lactalbumin) was dissolved in phosphate buffer at a concentration of 100 mg/ml. Then, trypsin (50 mg) was added. The mixture was allowed to react at 37 degrees C for 6 hours. After the reaction, centrifugation was carried out and the precipitate was removed. Then, ultrafiltration was carried out using membrane having a molecular weight cut off 10000. The active ingredient of the trypsin-hydrolyzed substances was fractionated by reverse phase HPLC, and the peptides derived from beta-lactoglobulin and alpha-lactalbumin were obtained. The peptides derived from beta-lactoglobulin and alpha-lactalbumin, were found to comprise sequences such as Ala-Leu-Pro-Met-His, Ala-Leu-Pro-Met-His-Ile-Arg, Phe-Lys-Ile-Asp-Ala-Leu-Asn-Glu, and Leu-Ala-His-Lys-Ala-Leu, Trp-Leu-Ala-His-Lys and Leu-Asp-Gln-Trp-Leu-Cys-Glu-Lys. The peptides when subjected to analysis, was found to possess TNF-alpha production inhibitory effect.(41 pages)

ACCESSION NUMBER: 2004-23477 BIOTECHDS
TITLE: New beta lactoglobulin, alpha lactalbumin or cow lactoferrin derived peptide, useful for treating systemic inflammatory response syndrome, retinopathy and rheumatoid arthritis; beta-lactoglobulin, alpha-lactalbumin or cattle **lactoferrin composition** for disease therapy
PATENT ASSIGNEE: MEIJI MILK PROD CO LTD
PATENT INFO: JP 2004196707 15 Jul 2004
APPLICATION INFO: JP 2002-367035 18 Dec 2002
PRIORITY INFO: JP 2002-367035 18 Dec 2002; JP 2002-367035 18 Dec 2002
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
OTHER SOURCE: WPI: 2004-521533 [50]

L3 ANSWER 14 OF 18 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Treating subject suffering from pain, involves administering **lactoferrin composition**, to provide improvement in pain in subjects; use of recombinant lactoferrin in a pharmaceutical composition for pain therapy
AN 2004-17265 BIOTECHDS
AB DERWENT ABSTRACT:

NOVELTY - Treating (M1) a subject suffering from pain, involves administering to the subject a **lactoferrin composition**, to provide an improvement in pain in the subject.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for modulating acute pain or chronic pain in a subject, involves administering **lactoferrin composition** to the subject to provide an improvement in acute pain or chronic pain in the subject.

WIDER DISCLOSURE - The following are disclosed: (1) **lactoferrin composition**; and (2) pharmaceutical composition comprising **lactoferrin composition**.

BIOTECHNOLOGY - Preferred Method: In (M1), the **lactoferrin composition** reduces the severity of the patient's pain. The **lactoferrin composition** is dispersed in a carrier. The lactoferrin is mammalian, preferably human or bovine lactoferrin. The lactoferrin is recombinant lactoferrin. The **lactoferrin**

composition comprises an N-terminal lactoferrin variant that lacks N-terminal glycine residue. The N-terminal lactoferrin variant comprises at least 1%-50% of the **lactoferrin composition**. (M1) further involves administering an antacid in conjunction with **lactoferrin composition**, and administering lactoferrin in a delayed release formulation. The lactoferrin release occurs in small intestine or large intestine. The **lactoferrin composition** reduces the production or activity of pro-inflammatory cytokines, or enhances the production or activity of cytokines (TNF-alpha). (M1) further involves administering a metal chelator such as EDTA or (ethylenebis(oxyethylenenitrilo))tetraacetic acid (EGTA), dispersed in a carrier. Preferably, the chelator is EDTA. (M1) further involves administering a **lactoferrin composition** in combination with pharmacological agent used to relieve pain. The pharmacological agent includes non-steroidal anti-inflammatory drugs (NSAIDS), opioid analgesics, second generation NSAIDs and anti-depressant drugs. (M1) further involves administering the **lactoferrin composition** in combination with non-pharmacological pain management technique chosen from acupuncture, acupressure, local anesthesia, regional anesthesia (spinal anesthesia), general anesthesia (intravenous anesthetics or opioid pump) and chiropractic.

ACTIVITY - Analgesic. The ability of recombinant human lactoferrin (rhLF) to reduce heat-induced pain in tail flick test in mice was done as follows. Mice received 1000 mg/kg rhLF or placebo orally 60 minutes prior to test (N = 5 per group). The tail flick test measured the time (max 15 seconds) required to elicit the radiation heat-induced tail-flick response in mice. Result showed that oral lactoferrin treatment significantly reduced pain in mice.

MECHANISM OF ACTION - Reduces production or activity of pro-inflammatory cytokines; Enhances production or activity of cytokines (claimed).

USE - (M1) is useful for treating a subject suffering from pain (acute or chronic pain) (claimed).

ADMINISTRATION - Administration of lactoferrin is orally, parenterally or topically, at a dose of 1 ng-100 g/day (preferably 0.1 g/10 g/day). The lactoferrin release occurs in small intestine or large intestine. The amount of EDTA that is administered as a chelator with the lactoferrin is 1 ng-1 g/day (all claimed).(30 pages)

ACCESSION NUMBER: 2004-17265 BIOTECHDS
TITLE: Treating subject suffering from pain, involves administering **lactoferrin composition**, to provide improvement in pain in subjects;
use of recombinant lactoferrin in a pharmaceutical composition for pain therapy
AUTHOR: VARADHACHARY A; PETRAK K
PATENT ASSIGNEE: AGENNIX INC
PATENT INFO: WO 2004054608 1 Jul 2004
APPLICATION INFO: WO 2003-US39358 11 Dec 2003
PRIORITY INFO: US 2003-498248 27 Aug 2003; US 2002-432937 12 Dec 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2004-488007 [46]

L3 ANSWER 15 OF 18 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Treating tissue or organ (e.g., kidney, heart, liver, lung or pancreas) transplant rejection in recipient involves administering a **lactoferrin composition** to the recipient to attenuate tissue or organ transplant rejection;
using recombinant lactoferrin for graft-versus-host-disease prevention and therapy and in tissue and organ transplantation
AN 2004-16861 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Treating a tissue or organ transplant rejection in a recipient involves administering to the recipient, a **lactoferrin composition** to attenuate the tissue or organ transplant rejection.

WIDER DISCLOSURE - (1) compositions comprising lactoferrin dispersed

in a carrier; (2) treating or preventing graft-versus-host-disease (GVHD) in a recipient by administering **lactoferrin composition** to the donor organ or donor tissue prior to transplantation into the recipient, or administering to the recipient, **lactoferrin composition**; and (3) treating, preventing or attenuating the severity of xenograft tissue or xenograft organ transplant rejection in a recipient by administering **lactoferrin composition** to xenograft donor.

BIOTECHNOLOGY - Preferred Method: The **lactoferrin composition** regulates T cell responses by inducing transplant tolerance in the recipient, and is dispersed in a carrier. The lactoferrin is mammalian lactoferrin e.g., human or bovine lactoferrin. Optionally, the lactoferrin is recombinant lactoferrin comprising an N-terminal lactoferrin variant, where the variant lacks at least the N-terminal glycine residue. The N-terminal variant comprises at least 1-50% pf the **lactoferrin composition**. The lactoferrin modulates the mucosal or systemic immune system in a subject by increasing the amount of lactoferrin in the gastrointestinal tract, where preferably lactoferrin stimulates interleukin-18 or MIP-3alpha in the gastrointestinal tract; regulates the activity of immune cells such as B lymphocytes and T lymphocytes (chosen from CD4+/CD3+, CD8+/CD3+ cells and natural killer (NK)-T cells), antigen-presenting cells, natural killer cells, macrophages and granulocytes; and regulates production or activity of pro-inflammatory cytokines. The method further comprises administering a metal chelator dispersed in a carrier.

ACTIVITY - Immunosuppressive.

MECHANISM OF ACTION - Reducer of allogenic immune responses in recipient; Regulator of T cell responses; Stimulator of interleukin-18 or MIP-3-alpha in the gastrointestinal tract; Regulator of activity of B and T lymphocytes, antigen-presenting cells, natural killer cells, macrophages and granulocytes; Regulator of production or activity of pro-inflammatory cytokines (all claimed). Heterotopic heart transplantation in 8-10 weeks old rats (BUF, donor to WF, recipient) was performed using standard microsurgical technique of end-to-side anastomoses to recipient aorta and vena cava. Graft survival was defined as the last day of transabdominally palpable cardiac contractions. Recipients were treated with either placebo or recombinant human lactoferrin (rhLF) (625 mg/Kg) for 14 days starting seven days prior to the transplant. The results showed that lactoferrin alone significantly extended cardiac allograft survival.

USE - For treating tissue (bone marrow or peripheral stem cells) or organ (kidney, heart, lung, liver, or pancreas) transplant rejection in the recipient (claimed).

ADMINISTRATION - The **lactoferrin composition** is administered orally, and the method further involves administering an antacid in conjunction with the **lactoferrin composition**, or administering the lactoferrin in delayed release formulation, where the release occurs in small and large intestine. Optionally, the **lactoferrin composition** is administered parenterally. The amount of the **lactoferrin composition** that is administered is 1 mg-20 g/day, preferably 0.1-5 g/day (all claimed). The metal chelator (EDTA) is administered in dosages of 1 ng-1 g/day.

ADVANTAGE - The method induces permanent allograft or xenograft acceptance and reducing the incidence of graft-versus-host-disease involved in bone marrow or peripheral stem cells transplantation. (38 pages)

ACCESSION NUMBER: 2004-16861 BIOTECHDS

TITLE: Treating tissue or organ (e.g., kidney, heart, liver, lung or pancreas) transplant rejection in recipient involves administering a **lactoferrin composition** to the recipient to attenuate tissue or organ transplant rejection;
using recombinant lactoferrin for graft-versus-host-disease prevention and therapy and in tissue and organ transplantation

AUTHOR: VARADHACHARY A; PERICLE F

PATENT ASSIGNEE: AGENNIX INC

PATENT INFO: WO 2004052305 24 Jun 2004

APPLICATION INFO: WO 2003-US39265 10 Dec 2003
PRIORITY INFO: US 2003-498338 27 Aug 2003; US 2002-432113 10 Dec 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2004-468695 [44]

L3 ANSWER 16 OF 18 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Treating a cardiovascular disease comprises administering to a subject an effective amount of a **lactoferrin composition** to provide an improvement in the cardiovascular disease in the subject; involving vector-mediated gene transfer and expression in host cell for use in gene therapy
AN 2004-16843 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Treating a cardiovascular disease comprises administering to a subject an effective amount of a **lactoferrin composition** to provide an improvement in the cardiovascular disease in the subject.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a method of modulating atherosclerosis in a subject comprising administering to the subject an effective amount of a **lactoferrin composition** to modulate atherosclerosis in the subject.

BIOTECHNOLOGY - Preferred Method: In treating a cardiovascular disease, the cardiovascular disease is atherosclerosis. The **lactoferrin composition** reduces levels of circulating total cholesterol, low-density lipoproteins (LDL), very low-density lipoproteins (VLDL), or triglycerides in the subject. The **lactoferrin composition** increases the levels of circulating high-density lipoproteins (HDL) in the subject. The **lactoferrin composition** reduces the levels of vascular inflammation, circulating C-reactive protein (CRP), proliferation of vascular smooth muscle cells, vascular spasm or vascular hyper-reactivity in the subject. The **lactoferrin composition** promotes endothelial integrity or healing in the subject. The **lactoferrin composition** is dispersed in a carrier. The lactoferrin is mammalian lactoferrin. The lactoferrin is human or bovine. The lactoferrin is recombinant lactoferrin. The **lactoferrin composition** comprises an N-terminal lactoferrin variant. The N-terminal lactoferrin variant lacks at least the N-terminal glycine residue. The N-terminal lactoferrin variant comprises at least 1% to at least 50% of the **lactoferrin composition**. The **lactoferrin composition** reduces the production or activity of pro-inflammatory cytokines. The method further comprises administering a **lactoferrin composition** in combination with an anti-cholesterol agent or an anti-inflammatory agent. The anti-cholesterol agent is selected from cholesterol absorption inhibitors, bile acid sequestrants, nicotinic acid, fibric acids and HMG-coA reductase inhibitors. The bile acid sequestrants are selected from cholestyramine, colestipol and colesevalam. The fibric acids are selected from gemfibrozil, fenofibrate and clofibrate. The HMG-coA reductase inhibitors are selected from lovastatin, pravastatin, simvastatin, fluvastatin, atorvastatin and cerivastatin. In modulating atherosclerosis in a subject, the modulating is reducing the incidence or severity of atherosclerosis in the subject.

ACTIVITY - Cardiant; Antiarteriosclerotic. No biological data given.

MECHANISM OF ACTION - Gene therapy; HMG-coA reductase inhibitor.

USE - The method is useful for treating a cardiovascular disease, e.g. atherosclerosis (claimed).

ADMINISTRATION - Dosage is 1 ng-20 g per day or 0.1-5 g per day. The **lactoferrin composition** is administered parenterally, e.g. subcutaneously, intramuscularly, intraperitoneally, intravenously, intraarterially, intramyocardially, transendocardially, transepicardially, or intrathecally, or orally (all claimed). (38 pages)

ACCESSION NUMBER: 2004-16843 BIOTECHDS
TITLE: Treating a cardiovascular disease comprises administering to a subject an effective amount of a **lactoferrin composition** to provide an improvement in the cardiovascular disease in the subject;

involving vector-mediated gene transfer and expression in host cell for use in gene therapy
AUTHOR: VARADHACHARY A; GLYNN P; WANG Y; ENGELMAYER J
PATENT ASSIGNEE: AGENNIX INC; VARADHACHARY A
PATENT INFO: WO 2004050037 17 Jun 2004
APPLICATION INFO: WO 2003-US38540 4 Dec 2003
PRIORITY INFO: US 2003-498337 27 Aug 2003; US 2002-430867 4 Dec 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2004-460986 [43]

L3 ANSWER 17 OF 18 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Use of a **lactoferrin composition** for the treatment of a respiratory disorder e.g. asthma, emphysema, bronchitis, chronic obstructive pulmonary disease;
human recombinant lactoferrin for allergic, non-allergic respiratory disorder, atopic asthma, non-atopic asthma, emphysema, bronchitis, chronic obstructive pulmonary disease, acute, chronic sinusitis or allergic rhinitis therapy
AN 2004-04222 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Treatment (M1) of a respiratory disorder comprising administrating a **lactoferrin composition** (c1), is new.

ACTIVITY - Antiasthmatic; Antiinflammatory; Antiallergic; Respiratory-Gen. Allergic sheep were treated with recombinant human lactoferrin (rhLF) and compared to their historic controls. Baseline bronchoalveolar lavage fluid (BAL) samples were taken from all animals and baseline dose response curves to aerosol carbachol were obtained in all sheep 1 - 3 days before the start of dosing. The sheep were pre-treated with oral rhLF (1 or 1.5 g) twice daily for 3 days prior to allergen challenge. Plasma samples were taken at both the time of 1st dose on day 1 and again on day 3 at the time of the second daily dose. On the challenge day (day 4) the test sheep received rhLF in dose of 1 g or 1.5 g 30 minutes before, 4 hours after and 24 hours after allergen challenge. BAL samples were again taken and additional plasma samples were also taken before and immediately after rhLF administration. On the challenge day (day 4) measurements of lung resistance (RL) were obtained before and then repeated 30 minutes after treatment and then the sheep were challenged with Ascaris suum allergen. Measurements of RL were obtained immediately after challenge, hourly form 1 - 8 hours after challenge. The RL (%) (for rhLF(1 g)/rhLF(1.5 g)/control) was 350/550/500 (at 0 hour), 0/0/0 (after 4 hours) and 0/20/100 (after 8 hours). The peak delayed increase in RL (%) was 76/74/0; the delayed airway hypersensitivity (%) was 100/88/0; the % increase in total inflammatory cells in the lungs was -/100/0 for rhLF(1 g)/rhLF(1.5 g)/control respectively.

MECHANISM OF ACTION - Interleukin-18 stimulator; Stimulator of production or activity of immune cells (preferably T lymphocytes and natural killer cells, especially CD4+, CD8+ and CD3+ cells); Granulocyte Macrophage Colony Stimulating Factor (GM-CSF) stimulator; Stimulator of production, maturation or activity of immune cells (preferably dendritic or other antigen representing cells); Inhibitor of production or activity of pro-inflammatory cytokines. The effect of oral administration of recombinant human lactoferrin (rhLF) on GM-CSF was tested in vivo. Mice (5 per group) were treated for 3 days daily with 300 mg/kg/day of rhLF. For a control, mice were only administered a pharmaceutical carrier. Twenty-four hours after administration of the LF or placebo for 3 days, animals were sacrificed and the small intestine tissue was removed for further analysis. Small intestinal epithelium was homogenized using a lysis buffer consisting of phosphate buffered saline (PBS), 1% Nonidet P-40, 0.5% sodium deoxycholate and 0.1% sodium dodecyl sulfate containing phenylmethylsulfonyl fluoride (10 microg/ml). Homogenate was centrifuged for 10 minutes and was tested for GM-CSF levels. The rhLF increased the production of a key immunostimulatory cytokine, GM-CSF in the small intestine compared to placebo (19.4%).

USE - The method is useful for the treatment of allergic or non-allergic respiratory disorder including atopic asthma, non-atopic

asthma, emphysema, bronchitis, chronic obstructive pulmonary disease, acute or chronic sinusitis, and allergic rhinitis (all claimed).

ADMINISTRATION - (c1) is administered in a dosage of 1 mg-1 g (preferably 10 mg-1 g) per day orally (claimed).

ADVANTAGE - (c1) enhances a mucosal immune response in the gastrointestinal tract; reduces the infiltration of inflammatory cells into the lung; reduces the delayed hypersensitivity associated with atopic or non-atopic asthma. The lactoferrin stimulates interleukin-18 in the gastrointestinal tract; stimulates the production, maturation or activity of immune cells (e.g. T lymphocytes (e.g. CD4+, CD8+, or CD3+ cells) or natural killer cells; stimulates GM-SCF in the gastrointestinal tract, thus stimulating the production, maturation or activity of immune cells (e.g. dendritic or other antigen presenting cells) and reduces the production or activity of pro-inflammatory cytokines.

EXAMPLE - No suitable example given. (28 pages)

ACCESSION NUMBER: 2004-04222 BIOTECHDS
TITLE: Use of a **lactoferrin composition** for the treatment of a respiratory disorder e.g. asthma, emphysema, bronchitis, chronic obstructive pulmonary disease; human recombinant lactoferrin for allergic, non-allergic respiratory disorder, atopic asthma, non-atopic asthma, emphysema, bronchitis, chronic obstructive pulmonary disease, acute, chronic sinusitis or allergic rhinitis therapy
AUTHOR: GLYNN P; VARADHACHARY A
PATENT ASSIGNEE: AGENNIX INC
PATENT INFO: WO 2003099207 4 Dec 2003
APPLICATION INFO: WO 2003-US15763 20 May 2003
PRIORITY INFO: US 2002-410645 13 Sep 2002; US 2002-383280 24 May 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2004-042695 [04]

L3 ANSWER 18 OF 18 HCAPLUS COPYRIGHT 2005 ACS on STN
TI **Lactoferrin composition** for inducing specific antibodies against lipid A of gram-negative microorganisms
AB Oral intake of lactoferrin induces production of specific IgG antibodies against lipid A in blood serum. The oral composition also shows immune responses against LPS blood serum type and against gram-neg. microorganisms of different strains. The composition added to infant milk formula produces no ill effects.
ACCESSION NUMBER: 2001:872964 HCAPLUS
DOCUMENT NUMBER: 136:5073
TITLE: **Lactoferrin composition** for inducing specific antibodies against lipid A of gram-negative microorganisms
INVENTOR(S): Nakamura, Yoshitaka; Takahashi, Takeshi; Yajima, Koji; Kuwata, Tamotsu
PATENT ASSIGNEE(S): Meiji Milk Products, Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
JP 2001333737	A2	20011204	JP 2001-85214		20010323
PRIORITY APPLN. INFO.:			JP 2000-84814	A	20000324

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(FILE 'HOME' ENTERED AT 15:53:22 ON 16 MAR 2005)

FILE 'MEDLINE, JAPIO, BIOSIS, WPIDS, JICST-EPLUS, BIOTECHDS, HCAPLUS, SCISEARCH, CEN, CEABA-VTB, BIOBUSINESS, EMBASE, DGENE' ENTERED AT

15:54:03 ON 16 MAR 2005
L1 1 S LACTOFERRIN AND (REDUCE CIRCULATING LEVELS OF CHOLESTEROL?)
L2 0 S LACTOFERRIN COMPOSITION ADJ2 ADMINISTRATION
L3 18 S LACTOFERRIN COMPOSITION

=> s lactoferrin
L4 26082 LACTOFERRIN

=> s l4 and (reduce vascular inflammation?)
L5 0 L4 AND (REDUCE VASCULAR INFLAMMATION?)

=> s heart disease and l4
L6 25 HEART DISEASE AND L4

=> d l6 ti abs ibib tot

L6 ANSWER 1 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Bovine **lactoferrin** reduces plasma triacylglycerol and NEFA accompanied by decreased hepatic cholesterol and triacylglycerol contents in rodents.
AB In the present study we examined whether oral administration of bovine **lactoferrin** (bLF) reduces plasma or hepatic triacylglycerol and cholesterol in mice. When bLF mixed with a standard commercial diet (10 g/kg) was given to mice for 4 weeks, plasma triacylglycerol and NEFA decreased, while plasma HDL-cholesterol levels increased (P 0.01). These changes in plasma lipid profiles were accompanied by significant decreases in hepatic cholesterol and triacylglycerol contents. When mice were fed a high-fat diet containing 300.0 g lard, 10.0 g cholesterol and 2.5 g bovine bile powder/kg for 4 weeks, bovine LF did not have any significant effects on plasma or hepatic cholesterol and triacylglycerol concentrations. Furthermore, bLF had no significant effects on faecal excretion of total bile acids in mice. Interestingly, bLF showed a suppressive effect on the lymphatic triacylglycerol absorption in chronically treated rats. We conclude that bLF has a beneficial effect on plasma cholesterol levels and retards hepatic lipid accumulation in mice fed a standard diet.
ACCESSION NUMBER: 2004:402668 BIOSIS
DOCUMENT NUMBER: PREV200400403547
TITLE: Bovine **lactoferrin** reduces plasma triacylglycerol and NEFA accompanied by decreased hepatic cholesterol and triacylglycerol contents in rodents.
AUTHOR(S): Takeuchi, Takashi; Shimizu, Hirohiko; Ando, Kunio; Harada, Etsumori [Reprint Author]
CORPORATE SOURCE: Fac AgrDept Vet Physiol, Tottori Univ, Tottori, 6800945, Japan
harada@muses.tottori-u.ac.jp
SOURCE: British Journal of Nutrition, (April 2004) Vol. 91, No. 4, pp. 533-538. print.
CODEN: BJNUAV. ISSN: 0007-1145.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Oct 2004
Last Updated on STN: 20 Oct 2004

L6 ANSWER 2 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Leucocyte depletion in cardiopulmonary bypass: A comparison of four strategies.
AB Leucocytes have been shown to play a fundamental role in the pathophysiology of inflammation. This prospective, randomized, controlled study was designed to identify the most advantageous leucocyte depletion technique in terms of reduction in systemic inflammatory response syndrome and myocardial ischaemia reperfusion injury associated with cardiopulmonary bypass (CPB). Forty consecutive patients undergoing elective coronary artery bypass graft (CABG) surgery were randomly allocated to one of four groups. The four groups consisted of a control group, a systemic leucocyte depletion (SLD) group, a cardioplegic leucocyte depletion (CLD) group, and a total leucocyte depletion (TLD) group. There were 10 patients in each group. **Lactoferrin** (marker of neutrophil activation) and troponin-I (marker of myocardial

ischaemia reperfusion injury) were measured at six time points: post induction, 5 min on CPB, 5 min before releasing the aortic crossclamp, 15 min after releasing the clamp and 1 and 24 hours after the discontinuation of CPB. Plasma **lactoferrin** levels increased rapidly in every group after the commencement of CPB, subsequently reached a peak after releasing the aortic crossclamp and gradually declined after the discontinuation of CPB. The lowest **lactoferrin** concentration was observed in the TLD (range 2.15-141.9 ng/mL) and CLD groups (7.469-114.6 ng/mL). Regarding myocardial injury, plasma cardiac troponin-I levels did not differ significantly between groups; but troponin-I concentrations rose dramatically after releasing the aortic crossclamp in all groups. Nevertheless, the CLD group had the lowest troponin-I level (1.37-5.55 ng/mL). In conclusion, it is believed that myocardial ischaemia is probably a major contributor to the inflammatory response. Although there is no clear statistical significance shown in this pilot study, the data tend to support the cardioplegic leucocyte depletion strategy as the optimal method for attenuating neutrophil activation and myocardial ischaemia reperfusion injury.

ACCESSION NUMBER: 2003:396640 BIOSIS
DOCUMENT NUMBER: PREV200300396640
TITLE: Leucocyte depletion in cardiopulmonary bypass: A comparison of four strategies.
AUTHOR(S): Samankatiwat, Piya [Reprint Author]; Samartzis, Ioannis; Lertsithichai, Panuwat; Stefanou, Demetrios; Punjabi, Prakash P.; Taylor, Kenneth M.; Gourlay, Terence
CORPORATE SOURCE: Department of Surgery, Faculty of Medicine, Mahidol University, Ramathibodi Hospital, Bangkok, Thailand
SOURCE: Perfusion (London), (April 2003) Vol. 18, No. 2, pp. 95-105. print.
ISSN: 0267-6591 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003

L6 ANSWER 3 OF 25 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI C-reactive protein and outcomes in unstable angina: Relationship to other markers of inflammation, vascular perturbation and necrosis.
ACCESSION NUMBER: 2001:369213 BIOSIS
DOCUMENT NUMBER: PREV200100369213
TITLE: C-reactive protein and outcomes in unstable angina: Relationship to other markers of inflammation, vascular perturbation and necrosis.
AUTHOR(S): Van Lente, F. [Reprint author]
CORPORATE SOURCE: Cleveland Clinic Foundation, Cleveland, OH, USA
SOURCE: Clinical Chemistry, (June, 2001) Vol. 47, No. S6, pp. A144. print.
Meeting Info.: 53rd Annual Meeting of the AACC/CSCC. Chicago, Illinois, USA. July 29-August 02, 2001. American Association for Clinical Chemistry.
CODEN: CLCHAU. ISSN: 0009-9147.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Aug 2001
Last Updated on STN: 19 Feb 2002

L6 ANSWER 4 OF 25 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Composition for supplementing nutritional deficiencies comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc, and copper.
AN 2004-634381 [61] WPIDS
CR 2004-440316 [41]
AB US2004166175 A UPAB: 20040923
NOVELTY - Composition for supplementing nutritional deficiencies (I) comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc and copper.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a

composition (II) comprising (mg) calcium (less than 160, preferably 90 - 110 mg), iron (more than 20, preferably 58.5 - 71.5) and copper (1.8 - 2.2).

ACTIVITY - Anabolic.

A study was undertaken to evaluate the effectiveness of the composition of the present invention in the treatment of patients. The objective of the study is to determine whether oral intake of the composition results in an improvement of the nutritional status of a patient in a physiologically stressful state. A double-blind, placebo controlled study was conducted over a six-month period. A total of 120 subjects (60 pregnant women entering the second trimester of pregnancy and 60 lactating women), aged 20 - 35 years, were chosen for the study. An initial assessment of the nutritional status of each woman was conducted utilizing methods such as the peroxide hemolysis test to assess Vitamin E deficiency, measurement of erythrocyte transketolase activity to determine thiamine levels, determination of erythrocyte glutathione reductase activity to assess riboflavin status, and high performance liquid chromatography to directly measure pyridoxine levels. The 120 subjects were separated into four separate groups of 30 women. In a first group comprising only pregnant women and in a second group comprising only lactating women, each subject was administered 2 caplets, daily, of (A1). In a third group comprising only pregnant women and in a fourth group comprising only lactating women, each subject was administered 2 placebo caplets, daily. No other nutritional supplements were taken by the subjects during the assessment period. An assessment of the nutritional status of each woman was conducted utilizing methods such as the peroxide hemolysis test to assess vitamin E deficiency, measurement of erythrocyte transketolase activity to determine thiamine levels, determination of erythrocyte glutathione reductase activity to assess riboflavin status, and high performance liquid chromatography to directly measure pyridoxine levels at one month intervals for a six month period. The data was evaluated using multiple linear regression analysis and a standard t-test. A statistically significant improvement in the nutritional status with respect to vitamin E, thiamine, riboflavin, and pyridoxine was observed in the treated subjects upon completion of the study over the controls.

MECHANISM OF ACTION - None given.

USE - For supplementing nutritional deficiencies to a patient (particularly a pregnant patient or a lactating patient) who is in a stressful state e.g. a disease state such as pulmonary disorder, a hematological/oncological disorder, a cancer, a disorder of the immune system, a cardiovascular disorder, a hepatic/biliary disorder, a disorder associated with pregnant females and a disorder associated with a fetus. The nutritional deficiencies are a result of elevated metabolic demand, increased plasma volume, decreased concentrations of nutrient-binding proteins (e.g. serum-ferritin, maltose-binding protein, **lactoferrin**, calmodulin, tocopheryl binding protein, riboflavin binding protein, retinol binding protein, transthyretin, high density lipoprotein-apolipoprotein A1, folic acid binding protein, and 25-hydroxyvitamin D binding protein) (all claimed). The disorders associated with pregnant females include osteomalacia and pre-eclampsia and disorders associated with the fetus include neural tube defects and various fetal abnormalities. The pulmonary disorder includes bronchitis, bronchiectasis, atelectasis, pneumonia, diseases caused by inorganic dusts, diseases caused by organic dusts, pulmonary fibrosis and pleurisy. The hematological/oncological disorder includes anemia, hemophilia, leukemia and lymphoma. The disorder of the immune system includes AIDS, AIDS-related complex and bacterial infection. The cardiovascular disorder includes arterial hypertension, orthostatic hypotension, arteriosclerosis, coronary artery disease, cardiomyopathy, arrhythmia, valvular **heart disease**, endocarditis, pericardial disease, cardiac tumor, aneurysm and peripheral vascular disorder. The hepatic/biliary disorder includes jaundice, hepatic steatosis, fibrosis, cirrhosis, hepatitis, hepatic granuloma, liver tumor, cholelithiasis, cholecystitis and choledocholithiasis.

ADVANTAGE - The compositions optimize good health, provide protection against poor nutrition and disease, provide specific nutrients before, during, and after the physiological processes of pregnancy or lactation, which has a profound, positive and comprehensive impact upon the overall

wellness of the developing and newborn child as well as the safety and health of the mother.

Dwg.0/0

ACCESSION NUMBER: 2004-634381 [61] WPIDS
CROSS REFERENCE: 2004-440316 [41]
DOC. NO. CPI: C2004-227792
TITLE: Composition for supplementing nutritional deficiencies comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc, and copper.
DERWENT CLASS: B04 B05 D13
INVENTOR(S): BALZER, C; GIORDANO, J A
PATENT ASSIGNEE(S): (BALZ-I) BALZER C; (GIOR-I) GIORDANO J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2004166175	A1	20040826	(200461)*		12

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2004166175	A1 Cont of	US 2002-315159	20021210
		US 2004-790027	20040302

PRIORITY APPLN. INFO: US 2002-315159 20021210; US 2004-790027 20040302

L6 ANSWER 5 OF 25 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Composition useful for supplementing nutritional deficiencies comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc and copper.
AN 2004-440316 [41] WPIDS
CR 2004-634381 [61]
AB US2004109901 A UPAB: 20041117
NOVELTY - A composition (C1) comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc and copper.

ACTIVITY - Respiratory-Gen.; Cytostatic; Antiinflammatory; Hepatotropic; Virucide; Vasotropic; Cardiant; Antiarrhythmic; Cardiovascular-Gen.; Antiarteriosclerotic; Hypertensive; Hypotensive; Anti-HIV; Hemostatic; Antianemic; Gynecological.

MECHANISM OF ACTION - None given.

USE - For supplementing nutritional deficiencies in a patient throughout the stressful states resulting from pregnancy, lactation, elevated metabolic demand, increased plasma volume, decreased concentrations of nutrient-binding proteins (e.g. serum-ferritin, maltose-binding protein, **lactoferrin**, calmodulin, tocopheryl binding protein, riboflavin binding protein, retinol binding protein, transthyretin, high density lipoprotein-apolipoprotein A1, folic acid binding protein and 25-hydroxyvitamin D binding protein) and any disease state (e.g. pulmonary disorder, hematological/oncological disorder, cancer, immune system disorder, cardiovascular disorder, hepatic/biliary disorder and disorder associated with pregnant females and fetus) (all claimed). Also useful for treating osteomalacia, pre-eclampsia, bronchitis, bronchiectasis, atelectasis, pneumonia, diseases caused by inorganic dusts, organic dusts, pulmonary fibrosis, pleurisy, anemia, hemophilia, leukemia, lymphoma, AIDS, HIV, arterial hypertension, orthostatic hypotension, arteriosclerosis, coronary artery disease, cardiomyopathy, arrhythmia, valvular **heart disease**, endocarditis, pericardial disease, cardiac tumor, aneurysm, peripheral vascular disorder, jaundice, hepatic steatosis, fibrosis, cirrhosis, hepatitis, hepatic granuloma, liver tumor, cholelithiasis, cholecystitis and choledocholithiasis.

ADVANTAGE - The composition provides nutritional supplement by preventing dietary deficiencies, and also protects against the development of disease.

Dwg.0/0
ACCESSION NUMBER: 2004-440316 [41] WPIDS
CROSS REFERENCE: 2004-634381 [61]
DOC. NO. CPI: C2004-164922
TITLE: Composition useful for supplementing nutritional deficiencies comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc and copper.
DERWENT CLASS: B05 D13
INVENTOR(S): BALZER, C; GIORDANO, J A; GIORDANO, J
PATENT ASSIGNEE(S): (BALZ-I) BALZER C; (GIOR-I) GIORDANO J A; (EVER-N) EVERETT LAB INC
COUNTRY COUNT: 107
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2004109901	A1	20040610	(200441)*		12
WO 2004052295	A2	20040624	(200441)	EN	
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW					
AU 2003296357	A1	20040630	(200472)		
US 6814983	B2	20041109	(200474)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2004109901	A1	US 2002-315159	20021210
WO 2004052295	A2	WO 2003-US39022	20031209
AU 2003296357	A1	AU 2003-296357	20031209
US 6814983	B2	US 2002-315159	20021210

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003296357	A1 Based on	WO 2004052295

PRIORITY APPLN. INFO: US 2002-315159 20021210

L6 ANSWER 6 OF 25 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Use of human apo-**lactoferrin** and peptides derivable from human **lactoferrin** for the production of composition useful for e.g. treating and preventing vascular disease.
AN 2003-712670 [67] WPIDS
AB WO2003072129 A UPAB: 20031017
NOVELTY - In the production of a composition, a substance containing human apo-**lactoferrin** and/or peptides derivable from human **lactoferrin** and/or its natural metabolites or equivalent analogs is used.
ACTIVITY - Antianginal; Cerebroprotective; Cardiant; Antiulcer; Antialopecia.
MECHANISM OF ACTION - VEGF165 induced angiogenesis inhibitor.
Lactoferrin, dissolved in saline, was given by tube feeding twice daily from Sunday afternoon (Day-1) to Friday afternoon (Day 4). Vehicle controls received saline by tube feeding. The angiogenesis treatment with VEGF was given intraperitoneally on Days 0 - 4 (twice daily). The results for test/control groups were vascularized area = 12.09 plus or minus 1.49/1.18 plus or minus 0.5, microvascular length = 1.465 plus or minus 0.077/0.28 plus or minus 0.04, and total microvascular length = 17.72 plus or minus 2.19/0.33 plus or minus 0.14 respectively. The results demonstrated that oral administration of apo-hLE significantly

enhanced the VEGF mediated angiogenic response.

USE - For treating and/or preventing vascular disease and/or states of tissue hypoperfusion (including impending or manifested stroke, ischemic **heart disease** e.g. angina pectoris or impending or manifested myocardial infarction), or peripheral artery occlusive disease with or without impending gangrene and/or state of depressed VEGF induced angiogenesis associated with peptic ulcer, leg ulcer or local or generalized hair loss) with hypoxia and/or ischemic consequences (claimed).

ADVANTAGE - The method is used in as an alternative to bypass surgery or any therapeutic angiogenesis options.

Dwg.0/0

ACCESSION NUMBER: 2003-712670 [67] WPIDS
DOC. NO. CPI: C2003-196034
TITLE: Use of human apo-**lactoferrin** and peptides derivable from human **lactoferrin** for the production of composition useful for e.g. treating and preventing vascular disease.
DERWENT CLASS: B04
INVENTOR(S): NORRBY, K
PATENT ASSIGNEE(S): (NORR-I) NORRBY K
COUNTRY COUNT: 29
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003072129	A1	20030904	(200367)*	EN	14
RW: AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LU MC NL PT SE SI SK TR					
W: AU JP US					
AU 2003210086	A1	20030909	(200428)		
EP 1478387	A1	20041124	(200477)	EN	
R: AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LU MC NL PT SE SI SK TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003072129	A1	WO 2003-SE329	20030227
AU 2003210086	A1	AU 2003-210086	20030227
EP 1478387	A1	EP 2003-743090	20030227
		WO 2003-SE329	20030227

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003210086	A1 Based on	WO 2003072129
EP 1478387	A1 Based on	WO 2003072129

PRIORITY APPLN. INFO: SE 2002-598 20020227

L6 ANSWER 7 OF 25 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Use of human apo-**lactoferrin** and peptides derivable from human **lactoferrin** for the production of composition useful for e.g. treating and preventing vascular disease;
human apo-**lactoferrin** for use in disease therapy
AN 2003-25156 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - In the production of a composition, a substance containing human apo-**lactoferrin** and/or peptides derivable from human **lactoferrin** and/or its natural metabolites or equivalent analogs is used.

ACTIVITY - Antianginal; Cerebroprotective; Cardiant; Antiulcer; Antialopecia.

MECHANISM OF ACTION - VEGF165 induced angiogenesis inhibitor. **Lactoferrin**, dissolved in saline, was given by tube feeding twice

daily from Sunday afternoon (Day-1) to Friday afternoon (Day 4). Vehicle controls received saline by tube feeding. The angiogenesis treatment with VEGF was given intraperitoneally on Days 0 - 4 (twice daily). The results for test/control groups were vascularized area = 12.09+/- 1.49/1.18+/- 0.5, microvascular length = 1.465+/- 0.077/0.28+/- 0.04, and total microvascular length = 17.72+/- 2.19/0.33+/- 0.14 respectively. The results demonstrated that oral administration of apo-hLE significantly enhanced the VEGF mediated angiogenic response.

USE - For treating and/or preventing vascular disease and/or states of tissue hypoperfusion (including impending or manifested stroke, ischemic **heart disease** e.g. angina pectoris or impending or manifested myocardial infarction), or peripheral artery occlusive disease with or without impending gangrene and/or state of depressed VEGF induced angiogenesis associated with peptic ulcer, leg ulcer or local or generalized hair loss) with hypoxia and/or ischemic consequences (claimed).

ADMINISTRATION - The route of administration is oral, parenteral, local or by inhalation. No dosage given.

ADVANTAGE - The method is used in as an alternative to bypass surgery or any therapeutic angiogenesis options.

EXAMPLE - No relevant example given. (14 pages)

ACCESSION NUMBER: 2003-25156 BIOTECHDS
TITLE: Use of human apo-**lactoferrin** and peptides derivable from human **lactoferrin** for the production of composition useful for e.g. treating and preventing vascular disease;
human apo-**lactoferrin** for use in disease therapy
AUTHOR: NORRBY K
PATENT ASSIGNEE: NORRBY K
PATENT INFO: WO 2003072129 4 Sep 2003
APPLICATION INFO: WO 2003-SE329 27 Feb 2003
PRIORITY INFO: SE 2002-598 27 Feb 2002; SE 2002-598 27 Feb 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-712670 [67]

L6 ANSWER 8 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Pharmaceutical composition for treatment of vascular disease or states of tissue hypoperfusion with hypoxic and/or ischemic consequences
AB Disclosed is the use of a substance selected from the group consisting of human apolactoferrin and/or peptides derivable from human **lactoferrin** and/or natural metabolites of human **lactoferrin** and/or functionally equivalent analogs of human apolactoferrin for the production of a pharmaceutical composition for treatment and/or prevention of a vascular disease and/or states of tissue hypoperfusion with hypoxic and/or ischemic consequences. Thus, oral or s.c. administration of apolactoferrin specifically enhanced the VEGF-mediated angiogenesis.

ACCESSION NUMBER: 2003:696760 HCAPLUS
DOCUMENT NUMBER: 139:219356
TITLE: Pharmaceutical composition for treatment of vascular disease or states of tissue hypoperfusion with hypoxic and/or ischemic consequences
INVENTOR(S): Norrby, Klas
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003072129	A1	20030904	WO 2003-SE329	20030227
W: AU, JP, US				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR				

EP 1478387 A1 20041124 EP 2003-743090 20030227
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, CY, TR, BG, CZ, EE, HU, SK
PRIORITY APPLN. INFO.: SE 2002-598 A 20020227
WO 2003-SE329 W 20030227
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
TI New ingredients from dairy foods
AB Dairy foods have traditionally been considered a source of nutrition, particularly high quality protein, calcium and other minerals, and vitamins. In the last few decades, milk and other dairy foods have been maligned due to their saturated fat and cholesterol contents and the belief that these constituents increase the risk of coronary **heart disease**. There are several studies, which indicate that dairy products may not potentiate atherosclerosis. In fact there are factors in milk that may actively protect against **heart disease** such as calcium, bioactive peptides, folic acid, vitamin B6, vitamin B12 and conjugated linoleic acid. A range of other activities has been demonstrated for dairy components. Milk proteins are an important source of bioactive peptides showing opioid and ACE-inhibitory activity. **Lactoferrin** has been shown to have a bifidus effect and antimicrobial activity. It also improves iron bioavailability. Glycomacropeptide, a-lactoglobulin, a-lactalbumin and casein phosphopeptides affect physiol. functions. The development of membrane technologies allows the fractionation of milk proteins to produce a range of products with potential impact on human health.
ACCESSION NUMBER: 2002:613228 HCAPLUS
TITLE: New ingredients from dairy foods
AUTHOR(S): Morgan, Wendy
CORPORATE SOURCE: N/A, North Sydney, NSW 2059, Australia
SOURCE: Abstracts of Papers, 224th ACS National Meeting, Boston, MA, United States, August 18-22, 2002 (2002), AGFD-041. American Chemical Society: Washington, D. C.
CODEN: 69CZPZ
DOCUMENT TYPE: Conference; Meeting Abstract
LANGUAGE: English

L6 ANSWER 10 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Formulations of tocopherols and methods of making and using them
AB Non-naturally-occurring compns. for use in amelioration of disruption of energy metabolism secondary to stress are described. The compns. comprise a tocopherol and/or a derivative thereof, and a synergist, and are particularly suited for use as nutritional supplements. Synergists include, but are not limited to, flavonoids and **lactoferrin** and/or derivs. thereof. Compns. comprising an optimized formulation comprising a tocopherol and an addnl. compound such as daidzein or biochanin A are also described. Methods of making these compns. and methods of ameliorating injury(ies) or disruption of energy metabolism secondary to stress, comprising administering such compns., are also disclosed. Various concns. of tocopherols and flavonoids were tested in vitro for the combined ability to ameliorate disruption of energy metabolism secondary to stress. For example, diosmin (3.3-100 μM) was not protective by itself, but was synergistic in that range with 10 μg/mL (±)-α-tocopherol, a concentration at which (±)-α-tocopherol was only slightly (about 15%) protective by itself. The combination of 100 μM diosmin and 100 μg/mL (±)-α-tocopherol greatly reduced cell death, providing about 70% protection against stress-induced cell death, indicating synergism between these components. A combinations of 100 μM diosmin and 11 μg/mL (±)-α-tocopherol was also synergistic.
ACCESSION NUMBER: 2002:570708 HCAPLUS
DOCUMENT NUMBER: 137:119700
TITLE: Formulations of tocopherols and methods of making and using them
INVENTOR(S): Miller, Guy; Brown, Lesley A.
PATENT ASSIGNEE(S): Galileo Laboratories, Inc., USA

SOURCE: U.S., 28 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6426362	B1	20020730	US 2000-684588	20001006
US 2003022818	A1	20030130	US 2002-188587	20020702

PRIORITY APPLN. INFO.: US 1999-158234P P 19991008
US 2000-684588 A1 20001006
REFERENCE COUNT: 95 THERE ARE 95 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Antitumor and anti-inflammatory properties of human **lactoferrin** and variants thereof
AB The invention provides compns. containing human **lactoferrin**, or **lactoferrin** variants deleted for one or more arginine residues in the amino-terminal region of the protein (i.e., in the first basic cluster), and methods of using the compns. The human **lactoferrin**, or **lactoferrin** variants, are useful for treatment of human diseases and conditions, including inflammation.
ACCESSION NUMBER: 1998:542975 HCAPLUS
DOCUMENT NUMBER: 129:166197
TITLE: Antitumor and anti-inflammatory properties of human **lactoferrin** and variants thereof
INVENTOR(S): Nuijens, Jan; Van Berkel, Patrick H. C.
PATENT ASSIGNEE(S): Pharming B.V., Neth.
SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9833509	A2	19980806	WO 1998-IB441	19980202
WO 9833509	A3	19990311		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9863076	A1	19980825	AU 1998-63076	19980202
EP 1017407	A2	20000712	EP 1998-907139	19980202
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 6333311	B1	20011225	US 1998-17043	19980202
JP 2002515893	T2	20020528	JP 1998-532679	19980202

PRIORITY APPLN. INFO.: US 1997-36859P P 19970203
US 1998-17043 A 19980202
WO 1998-IB441 W 19980202

L6 ANSWER 12 OF 25 HCAPLUS COPYRIGHT 2005 ACS on STN
TI The erythrocyte as instigator of inflammation. Generation of amidated C3 by erythrocyte adenosine deaminase
AB Myocardial ischemia is characterized by the liberation of adenosine and by complement-mediated inflammation. The authors have reported that amidated complement C3, formed when ammonia disrupts the thiolester bond of C3, serves as an alternative pathway convertase, generates C5b-9, and stimulates phagocytic oxidative metabolism Here, it was investigated whether

the deamination of adenosine by adenosine deaminase in hematopoietic cells might liberate sufficient ammonia to form amidated C3 and thereby trigger complement-mediated inflammation at ischemic sites. In the presence of 4 mM adenosine, NH3 production per erythrocyte (RBC) was equal to that per neutrophil (PMN). Because RBC outnumber PMN in normal blood by a thousand-fold, RBC are the major source of NH3 production in the presence of adenosine. NH3 production derived only from the deamination of adenosine by the enzyme adenosine deaminase and was abolished by 0.4 μM 2'-deoxy-coformycin, a specific inhibitor of adenosine deaminase. When purified human C3 was incubated with 5 + 108 human RBC in the presence of adenosine, disruption of the C3 thiolester increased more than two-fold over that measd. in C3 incubated with buffer, or in C3 incubated with RBC. The formation of amidated C3 was abolished by the preincubation of RBC with 2'-deoxycoformycin. Amidated C3 elicited release of superoxide, myeloperoxidase, and **lactoferrin** from PMN. Thus, the formation of amidated C3 by RBC deamination of adenosine triggers a cascade of complement-mediated inflammatory reactions.

ACCESSION NUMBER: 1989:532298 HCAPLUS
DOCUMENT NUMBER: 111:132298
TITLE: The erythrocyte as instigator of inflammation. Generation of amidated C3 by erythrocyte adenosine deaminase
AUTHOR(S): Hostetter Margaret K.; Johnson, George M.
CORPORATE SOURCE: Med. Sch., Univ. Minnesota, Minneapolis, MN, 55455, USA
SOURCE: Journal of Clinical Investigation (1989), 84(2), 665-71
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English

L6 ANSWER 13 OF 25 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Phenolics, their antioxidant and antimicrobial activity in dark germinated fenugreek sprouts in response to peptide and phytochemical elicitors

AB The phenylpropanoid pathway (PPP) was stimulated in fenugreek sprouts through the pentose phosphate and shikimate pathway, by natural elicitors such as Fish Protein Hydrolysates (FPH), **Lactoferrin** (LF) and Oregano Extract (OE). Among treatments 0.5 ml/L FPH elicited fenugreek sprouts had the highest phenolic content of 0.75 mg/g FW on day 3 of germination which was approximately 25% higher than control on the same day. The antioxidant activity estimated by beta-carotene assay was highest for LF and OE elicited sprouts on day 2 and 4, respectively with an antioxidant protection factor (APF) of 1.47 for both. In all treatments and control, higher antioxidant activity was observed during early germination, which correlates to higher phenolic content, suggesting that initially phenolics are antioxidant in nature. This increased activity also correlates with high guaiacol peroxidase (GPX) activity indicating that the polymerized phenolics required for lignification with growth have antioxidant function. The antioxidant activity as estimated by beta-carotene and 1,1,-diphenyl-2-picryl hydrazyl (DPPH) assays indicate that fenugreek sprout extract can quench the superoxide free radical and also possibly scavenge the hydrogen peroxide generated in the reaction mix. OE elicited the highest levo dihydroxy phenylalanine (L-DOPA) synthesis of 1.59 mg/g FW, followed by FPH with 1.56 mg/g FW and LF 1.5 mg/g FW all on day 2 which was 24.5%, 23% and 20% higher than control, respectively. Higher L-DOPA content was observed in the elicited fenugreek sprouts during early germination, correlating to high phenolics and antioxidant activity, suggesting that L-DOPA also contributes to the high antioxidant activity. The glucose-6-phosphate dehydrogenase (G6PDH) activity was higher during early germination (day 1-4) and gradually decreased during later stages (day 5-8) for all treatments and control. The early increase is possibly due to the carbohydrate mobilization from the cotyledons directed towards the high nutrient requirements of the growing sprout. As mobilization occurred, an allosteric feedback inhibition by sugar-phosphates is suggested, as lower G6PDH activity was observed on days 6-8. The elevated levels of GPX during early germination coincide with the higher phenolic synthesis; SOD activity and antioxidant

activity suggests the elevated production and quenching of reactive oxygen species by elicitation. High antimicrobial activity against peptic ulcer-linked Helicobacter pylori was observed in the fenugreek sprout extract from control and LF treatments only. We hypothesized that in fenugreek sprouts, simple free phenolics that are less polymerized have more antimicrobial function.

ACCESSION NUMBER: 2004:873382 SCISEARCH
THE GENUINE ARTICLE: 859EW
TITLE: Phenolics, their antioxidant and antimicrobial activity in dark germinated fenugreek sprouts in response to peptide and phytochemical elicitors
AUTHOR: Randhir R; Lin Y T; Shetty K (Reprint)
CORPORATE SOURCE: Univ Massachusetts, Chenoweth Lab, Dept Food Sci, Amherst, MA 01003 USA (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: ASIA PACIFIC JOURNAL OF CLINICAL NUTRITION, (SEP-OCT 2004) Vol. 13, No. 3, pp. 295-307.
Publisher: H E C PRESS, HEALTHY EATING CLUB PTY LTD, EMERALD HILL CLINIC 157 CLARENDON ST, SOUTHBANK, VIC 3006, AUSTRALIA.
ISSN: 0964-7058.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 60
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L6 ANSWER 14 OF 25 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Elimination of proinflammatory cytokines in pediatric cardiac surgery: Analysis of ultrafiltration method and filter type
AB Objective: This study was undertaken to assess whether different filter types or ultrafiltration methods influence inflammatory markers in pediatric cardiac surgery.

Methods: Forty-one children younger than 5 years were prospectively randomized to groups A (polyamid filter with conventional ultrafiltration), B (polyamid filter with modified ultrafiltration), C (polysulfon filter with conventional ultrafiltration), and D (polysulfon filter with modified ultrafiltration). Interleukin 6, interleukin 10, tumor necrosis factor, terminal complement complex, and **lactoferrin** were measured before the operation (TO), before rewarming (T1), after ultrafiltration (T2), at 6 (T3) and 18 hours (T4) after the operation, and in the ultrafiltrate.

Results: All markers changed with both ultrafiltration methods, both filter types, and in all groups (except tumor necrosis factor) along the TO to T4 observation time ($P < .0001$). Their patterns of changes were different for terminal complement complex, with less decrease after use of the polysulfon filter ($P < .05$), and among groups A through D for interleukin 6 ($P = .01$), with more decrease in group C than group A ($P < .02$). Interleukin 10 decreased with the polyamid filter ($P < .001$) but not with the polysulfon filter. In the ultrafiltrate, tumor necrosis factor was higher with the polysulfon filter than the polyamid filter (6.8 +/- 5 pg/mL vs 4.0 +/- 3.7 pg/mL, $P < .05$). The ultrafiltrate/plasma ratio of interleukin 6 was higher with conventional ultrafiltration than modified ultrafiltration (0.018 +/- 0.017 vs 0.004 +/- 0.007, $P < .005$).

Conclusions: The polysulfon filter showed a filtration profile for inflammatory mediators superior to that of the polyamid filter for interleukin 6, tumor necrosis factor, and interleukin 10. Interleukin 6 was most efficiently removed by conventional ultrafiltration with a polysulfon filter, and tumor necrosis factor was best removed by modified ultrafiltration with a polysulfon filter, whereas other inflammatory mediators were not influenced by filter type or ultrafiltration method. Therefore combined conventional and modified ultrafiltration with a polysulfon filter may currently be the most effective strategy for removing inflammatory mediators in pediatric heart surgery.

ACCESSION NUMBER: 2004:558616 SCISEARCH
THE GENUINE ARTICLE: 827JN
TITLE: Elimination of proinflammatory cytokines in pediatric cardiac surgery: Analysis of ultrafiltration method and

filter type
AUTHOR: Berdat P A (Reprint); Eichenberger E; Ebell J; Pfammatter J P; Pavlovic M; Zobrist C; Gygax E; Nydegger U; Carrel T
CORPORATE SOURCE: Univ Hosp Bern, Swiss Cardiovasc Ctr Berne, Cardiovasc Surg Clin, CH-3010 Bern, Switzerland (Reprint); Univ Hosp Bern, Div Pediat Cardiol, CH-3010 Bern, Switzerland; Univ Hosp Bern, Div Cardiovasc Anesthesiol, CH-3010 Bern, Switzerland
COUNTRY OF AUTHOR: Switzerland
SOURCE: JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY, (JUN 2004) Vol. 127, No. 6, pp. 1688-1696.
Publisher: MOSBY, INC, 11830 WESTLINE INDUSTRIAL DR, ST LOUIS, MO 63146-3318 USA.
ISSN: 0022-5223.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 25
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L6 ANSWER 15 OF 25 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Neutrophil activation and C-reactive protein concentration in preeclampsia
AB Preeclamptic pregnancies seem to be associated with a higher extent of inflammation compared with normal ones. We intended to test this proposal and also to clarify the contribution of some variables in such inflammatory process. We measured total and differential leukocyte count, serum C-reactive protein (CRP), and plasma levels of **lactoferrin**, elastase, and granulocyte-macrophage colony-stimulating factor (GMCSF). Uric acid was also evaluated and used as an indicator of the severity of the disease. A cross-sectional study was performed by evaluating healthy and preeclamptic women in the third trimester of gestation (n = 67 and n = 51, respectively) and 24 to 48 h postpartum (n = 32 and n = 26, respectively). When comparing the third trimester of normal and preeclamptic pregnancies, we found significantly higher levels of uric acid, CRP, and elastase, and a significantly higher elastase to neutrophil ratio in the pathologic group. However, for CRP, statistical significance was lost after adjustment for maternal weight. No significant differences were found in total leukocyte count, plasma levels of GM-CSF, and **lactoferrin** between groups. In preeclampsia, a significant positive correlation was found between elastase and **lactoferrin** and these neutrophil activation products correlated positively with uric acid level. Considering the analysis of all variables in the postpartum period, only CRP and uric acid levels were significantly elevated in the pathologic group. However, CRP differences obtained in the puerperium seem to be influenced by the increased number of dystocic deliveries in the preeclamptic group. In conclusion, our data suggest that inflammation is further pronounced in preeclampsia and that the extent of neutrophil activation correlates with the severity of this syndrome.
ACCESSION NUMBER: 2003:606328 SCISEARCH
THE GENUINE ARTICLE: 699FC
TITLE: Neutrophil activation and C-reactive protein concentration in preeclampsia
AUTHOR: Belo L (Reprint); Santos-Silva A; Caslake M; Cooney J; Pereira-Leite L; Quintanilha A; Rebelo I
CORPORATE SOURCE: Univ Porto, Fac Pharm, Dept Biochem, Rua Campo Alegre 823, P-4050047 Oporto, Portugal (Reprint); Univ Porto, Fac Pharm, Dept Biochem, P-4050047 Oporto, Portugal; Univ Porto, Inst Mol & Cell Biol, P-4100 Oporto, Portugal; Univ Glasgow, Glasgow Royal Infirm, NHS Trust, Dept Pathol Biochem, Glasgow, Lanark, Scotland; Hosp Sao Joao, Porto Med Sch, Dept Obstet & Gynaecol, Oporto, Portugal; Univ Porto, Inst Biomed Sci Abel Salazar, P-4100 Oporto, Portugal
COUNTRY OF AUTHOR: Portugal; Scotland
SOURCE: HYPERTENSION IN PREGNANCY, (JUL 2003) Vol. 22, No. 2, pp. 129-141.
Publisher: MARCEL DEKKER INC, 270 MADISON AVE, NEW YORK, NY 10016 USA.

ISSN: 1064-1955.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 43
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L6 ANSWER 16 OF 25 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Improvement of long-standing iron-deficiency anemia in adults after eradication of Helicobacter pylori infection
AB We report two cases of long-standing iron-deficiency anemia in premenopausal women that improved after eradication of H. pylori infection. There were no ulcerations or hemorrhagic lesions in the gastrointestinal tract and no bleeding focus in gynecological organs. Both cases showed H. pylori infection in the stomach and gastric atrophy. After successful eradication of H. pylori infection, the iron-deficiency anemia in both patients dramatically improved, and neither patient suffered from anemia for about 2 years. The cure of H. pylori infection is an optional treatment for iron-deficiency anemia in one fraction of the patients.
ACCESSION NUMBER: 2002:507770 SCISEARCH
THE GENUINE ARTICLE: 561VJ
TITLE: Improvement of long-standing iron-deficiency anemia in adults after eradication of Helicobacter pylori infection
AUTHOR: Sugiyama T (Reprint); Tsuchida M; Yokota K; Shimodan M; Asaka M
CORPORATE SOURCE: Hokkaido Univ, Grad Sch Med, Dept Gastroenterol, Kita Ku, Kita 15, Nishi 7, Sapporo, Hokkaido 0608638, Japan (Reprint); Hokkaido Univ, Grad Sch Med, Dept Gastroenterol, Kita Ku, Sapporo, Hokkaido 0608638, Japan
COUNTRY OF AUTHOR: Japan
SOURCE: INTERNAL MEDICINE, (JUN 2002) Vol. 41, No. 6, pp. 491-494. Publisher: JAPAN SOC INTERNAL MEDICINE, 34-3 3-CHOME HONGO BUNKYO-KU, TOKYO, 113, JAPAN.
ISSN: 0918-2918.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 23
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L6 ANSWER 17 OF 25 CEN COPYRIGHT 2001 ACS on STN

TI BIG FIRMS EYE NUTRACEUTICALS
Polyunsaturated fatty acids and other nutritional ingredients are pursued by the chemical industry's leading companies
ACCESSION NUMBER: 2000:2447 CEN
TITLE: BIG FIRMS EYE NUTRACEUTICALS
Polyunsaturated fatty acids and other nutritional ingredients are pursued by the chemical industry's leading companies
SOURCE: Chemical & Engineering News, (25 Sep 2000) Vol. 78, No. 39, pp. 21.
CODEN: CENEAR, ISSN: 0009-2347.
PUBLISHER: American Chemical Society
LANGUAGE: English
WORD COUNT: 2428

L6 ANSWER 18 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI CXC-chemokine stimulation of neutrophils correlates with plasma levels of myeloperoxidase and **lactoferrin** and contributes to clinical outcome after pediatric cardiac surgery.
AB Several CXC-chemokines, of which interleukin (IL)-S is the prototype, are potent neutrophil chemotactic and activating cytokines, inducing the secretion of granule proteins and the generation of reactive oxygen intermediates that may cause tissue damage and amplify inflammatory responses. Here, we investigated whether chemokines play a key role in the inflammatory process following cardiac surgery with cardiopulmonary bypass; (CPB) in children. We performed an observational prospective

clinical study of 40 pediatric patients before, during, and after open heart surgery with CPB. Plasma levels of chemokines, myeloperoxidase (MPO), and **lactoferrin** were measured by immunoassays. Cell surface receptors were detected by flow cytometry. Plasma levels of IL-8 were increased after CPB, correlating strongly with a reduction of expression of the CXC-chemokine receptors (CXCR) 1 and 2 on neutrophils indicating in vivo activation of neutrophils by IL-8. Other CXC-chemokines with Glu-Leu-Arg motif showed no correlation with CXCR1 or CXCR2 expression. Two components of neutrophilic granules, MPO and **lactoferrin**, were strongly elevated postoperatively, and the levels of both were correlated with IL-8. Levels of monocyte chemoattractant protein (MCP)-1 were increased post-operatively, correlating with a reduction of CCR2 expression and an increase of CD11b expression on monocytes, suggesting monocyte activation by MCP-1. The early postoperative course was complicated in patients with an increase of these inflammatory parameters. Impaired cardiovascular function correlated with increased levels of IL-8 and activation of neutrophils and was most prominent in patients with a long time on CPB and in those with cyanotic heart lesions. In conclusion, MCP-1 is involved in the regulation of chemotaxis and function of monocytes during and early after the end of CPB. Activation of neutrophils and down-regulation of CXCR1 and CXCR2 were predominantly caused by IL-8. This activation implies release of components of neutrophilic granules and correlates with the need for inotropic support.

ACCESSION NUMBER: 2004515082 EMBASE
TITLE: CXC-chemokine stimulation of neutrophils correlates with plasma levels of myeloperoxidase and **lactoferrin** and contributes to clinical outcome after pediatric cardiac surgery.
AUTHOR: Gessler P.; Pretre R.; Hohl V.; Rousson V.; Fischer J.; Dahinden C.
CORPORATE SOURCE: Dr. P. Gessler, University Children's Hospital, Steinwiesstrasse 75, CH 8032 Zurich, Switzerland. peter.gessler@kispi.unizh.ch
SOURCE: Shock, (2004) 22/6 (513-520).
Refs: 40
ISSN: 1073-2322 CODEN: SAGUAI
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 19 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Cellular effects of common nutraceuticals and natural food substances.
ACCESSION NUMBER: 2004053658 EMBASE
TITLE: Cellular effects of common nutraceuticals and natural food substances.
AUTHOR: Mandelker L.; Wynn S.
CORPORATE SOURCE: L. Mandelker, Community Veterinary Hospital, 1631 W. Bay Drive, Largo, FL 33770, United States. lestervet2@aol.com
SOURCE: Veterinary Clinics of North America - Small Animal Practice, (2004) 34/1 (339-353).
ISSN: 0195-5616 CODEN: VCNAA6
PUBLISHER IDENT.: S 0195-5616(03)00135-9
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
052 Toxicology
LANGUAGE: English

L6 ANSWER 20 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Intracellular renin and the nature of intracrine enzymes.

AB Recently, the binding of renin and prorenin to cellular receptors with the subsequent generation of second messengers and the production of physiological effects has been demonstrated. In addition, the internalization of prorenin by target cells has been associated with increased cellular synthesis of angiotensin and cardiac pathology. Also, a renin transcript lacking the sequences encoding a secretory signal has been reported, and this transcript appears to produce a renin that acts in the cell that synthesized it. Some years ago, we coined the term intracrine for a peptide hormone or factor that acts in the intracellular space either after internalization or retention in its cell of synthesis. Thus defined, a wide variety of peptides display intracrine functionality, including hormones, growth factors, transcription factors, and enzymes. For example, considerable evidence indicates that angiotensin II is an intracrine. Also, general principles of intracrine functionality have been developed. Thus, recent evidence demonstrates that the prorenin/renin molecule is an intracrine enzyme. Here, the actions of intracrine enzymes (angiogenin, phosphoglucose isomerase, phospholipase A2, granzyme A and B, thioredoxin, platelet-derived endothelial growth factor, and serine protease inhibitors) are reviewed. The relation of prorenin/renin to other intracrine enzymes, and to intracrines in general, is discussed.

ACCESSION NUMBER: 2003317098 EMBASE
TITLE: Intracellular renin and the nature of intracrine enzymes.
AUTHOR: Re R.N.
CORPORATE SOURCE: Dr. R.N. Re, Research Division, Ochsner Clinic Foundation, 1514 Jefferson Highway, New Orleans, LA 70121, United States. rre@ochsner.org
SOURCE: Hypertension, (1 Aug 2003) 42/2 (117-122).
Refs: 75
ISSN: 0194-911X CODEN: HPRTDN
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 21 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Duraflo II coating of cardiopulmonary bypass circuits reduces complement activation, but does not affect the release of granulocyte enzymes in fully heparinized patients: A European multicentre study.

AB Objective: This study was carried out to: (a) compare complement and granulocyte activation during cardiac operations in patients operated with cardiopulmonary bypass coated with heparin by the Duraflo II method, with activation in patients operated with uncoated circuits; and (b) relate complement, and granulocyte activation to selected adverse effects, Methods: In a multicentre study among Rikshospitalet, Ullevaal Hospital in Norway and Uppsala University Hospital in Sweden, plasma concentrations of the complement activation products C4b/iC4b/C4e (C4bc), C3b/iC3b/C3e (C3bc), the terminal SC5b-9 complement complex (TCC), and the granulocyte proteins myeloperoxidase and **lactoferrin** were assessed in two groups of patients undergoing aortocoronary bypass. Seventy-six patients underwent surgery operated with circuits coated by the Duraflo II heparin coating and 75 with uncoated circuits. The same amount of systemic heparin was administered to all patients. Results: In both groups a significant increase in C4bc was first seen by the end of operation, from 86.7 ± 12.5 to 273.0 ± 277.4 nM in controls and from 86.9 ± 18.5 to 320.2 ± 190.5 nM in the control group, confirming previous documentation that the classical pathway is not activated during CPB, but as a consequence of protamin administration. The formation of C4bc did not differ significantly between the two groups. In the uncoated group the C3bc

concentration increased from 124.0 ± 15.3 to a maximum of 1176.1 ± 64.7 nM ($P < 0.01$) and in the coated group it increased from 129.8 ± 16.1 to a maximum of 1019.4 ± 54.9 nM ($P < 0.01$) during CPB. Summary values but not peak values differed significantly between the groups. In the uncoated group the TCC concentration increased from 0.52 ± 0.03 to a maximum value of 8.09 ± 0.57 AU/ml ($P < 0.01$) while in the coated group the TCC concentration increased from a baseline of 0.53 ± 0.03 to a peak value of 5.2 ± 0.24 AU/ml ($P < 0.01$). The difference between the peak values was statistically significant ($P = 0.00002$). In both groups a significant increase in myeloperoxidase and **lactoferrin** release was observed by the end of operation. There was no difference in myeloperoxidase or **lactoferrin** release between the two groups. TCC levels were compared to the occurrence of perioperative infarction, development of lung or renal failure, postoperative bleeding, time on ventilator and days in hospital. Three patients developed perioperative infarction; the peak levels of TCC were significantly higher in these patients than in the 148 patients that did not develop infarction. The reduction in TCC formation in the heparin-coated group was not associated with differences in any of the other clinical parameters. Few adverse effects occurred in the study. The peak values of C3bc were higher in the patients needing inotropic support than in those who did not, the relevance of this finding remains uncertain. Conclusion: It is concluded that the Duraflo II heparin coating reduces complement activation, particularly TCC formation, during CPB, but not the release of specific neutrophil granule enzymes. No certain correlation was established between complement and granulocyte activation and clinical outcome.

ACCESSION NUMBER: 97068280 EMBASE
DOCUMENT NUMBER: 1997068280
TITLE: Duraflo II coating of cardiopulmonary bypass circuits reduces complement activation, but does not affect the release of granulocyte enzymes in fully heparinized patients: A European multicentre study.
AUTHOR: Fosse E.; Thelin S.; Svennevig J.L.; Jansen P.; Mollnes T.E.; Hack E.; Venge P.; Moen O.; Brockmeier V.; Dregelid E.; Halden E.; Hagman L.; Videm V.; Pedersen T.; Mohr B.
CORPORATE SOURCE: E. Fosse, Dept.of Thor.Surgery/Anaesthesiology, Ullevaal Hospital, Oslo, Norway
SOURCE: European Journal of Cardio-thoracic Surgery, (1997) 11/2 (320-327).
Refs: 27
ISSN: 1010-7940 CODEN: EJCSE7
PUBLISHER IDENT.: S 1010-7940(96)01062-7
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
018 Cardiovascular Diseases and Cardiovascular Surgery
025 Hematology
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 22 OF 25 EMBASE
on STN
TI Attenuation of changes in leukocyte surface markers and complement activation with heparin-coated cardiopulmonary bypass.
AB Background. The inflammatory response induced by cardiopulmonary bypass can result in severe organ dysfunction in some patients. This postperfusion response is caused mainly by contact between blood and the foreign surface of the cardiopulmonary bypass equipment and includes adhesion of leukocytes to vascular endothelium, which precedes a series of events that mediate inflammatory damage to tissues. Methods. Low-risk patients accepted for coronary artery bypass grafting were randomized to operation with the cardiopulmonary bypass surface either completely heparin coated (Duraflo II) or uncoated. There were 12 patients in each group. Blood plasma sampled during cardiopulmonary bypass was analyzed for complement activation (C3bc and terminal SC5b-9 complement complex) and neutrophil activation (**lactoferrin** and myeloperoxidase). In addition, neutrophils, monocytes, and platelets were counted, and the

expression of surface markers on the neutrophils and monocytes (complement receptor [CR] 1, CR3, CR4, and L-selectin) and on the platelets (p-selectin and CD41) was quantified with flow cytometry. Results. Clinical and surgical results were similar in both groups. In the group with the heparin-coated surface, the formation of the terminal SC5b-9 complement complex was significantly reduced, and the counts of circulating leukocytes and platelets were significantly less reduced initially but were higher at the end of cardiopulmonary bypass compared with baseline. Also, the expression of CR1, CR3, and CR4 was significantly less upregulated and the L-selectin, significantly less downregulated on monocytes and neutrophils. Conclusions. We conclude that heparin coating reduces complement activation and attenuates the leukocyte integrin and selectin response that occurs when uncoated circuits are used.

ACCESSION NUMBER: 97027337 EMBASE
DOCUMENT NUMBER: 1997027337
TITLE: Attenuation of changes in leukocyte surface markers and complement activation with heparin-coated cardiopulmonary bypass.
AUTHOR: Moen O.; Hogasen K.; Fosse E.; Dregelid E.; Brockmeier V.; Venge P.; Harboe M.; Mollnes T.E.
CORPORATE SOURCE: Dr. O. Moen, Department of Cardiothoracic Surgery, Ulleval Hospital, N-0407 Oslo, Norway
SOURCE: Annals of Thoracic Surgery, (1997) 63/1 (105-111).
Refs: 25
ISSN: 0003-4975 CODEN: ATHSAK
PUBLISHER IDENT.: S 0003-4975(96)00743-6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 009 Surgery
015 Chest Diseases, Thoracic Surgery and Tuberculosis
018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 23 OF 25 EMBASE
on STN
TI Myocardial neutrophil sequestration and activation related to the reperfusion of human heart during coronary artery surgery.
AB Objective: The aim was to determine if neutrophils are activated and sequestered as they pass through postischaemic human myocardium. Methods: The occurrence of neutrophil activation during the reperfusion of the ischaemic myocardium was investigated in 16 selected patients undergoing coronary artery bypass surgery. Neutrophils were counted and elastase and **lactoferrin** released into the plasma were measured simultaneously in myocardial venous blood and in peripheral venous blood, before aortic cross clamping (T0), and two (T1), 10 (T2), and 20 (T3) min after unclamping. Results: At T0, no statistically significant difference was noted between peripheral and myocardial blood with respect to the three variables studied. Reperfusion was associated with a significantly lower neutrophil count in myocardial blood compared to peripheral blood ($p<0.001$), suggesting that neutrophils were trapped within the myocardium during reperfusion. In addition, levels of elastase (T1, T2, and T3), and **lactoferrin** (T1) were significantly higher in myocardial blood as compared to peripheral blood ($p < 0.001$), suggesting that activated neutrophils released their granular content into the plasma milieu. Conclusion: We provide evidence consistent with local neutrophil activation during myocardial reperfusion in patients undergoing coronary artery bypass surgery, in addition to the well described systemic activation related to cardiopulmonary bypass.

ACCESSION NUMBER: 94262503 EMBASE
DOCUMENT NUMBER: 1994262503
TITLE: Myocardial neutrophil sequestration and activation related to the reperfusion of human heart during coronary artery surgery.
AUTHOR: Farah B.; Vuillemenot A.; Lecompte T.; Bara L.; Pasquier

C.; Jebara V.; Carpentier A.; Fabiani J.
CORPORATE SOURCE: Serv. de Chirurgie Cardio-Vasculaire, Hopital Broussais, 96 rue Didot,75014 Paris, France
SOURCE: Cardiovascular Research, (1994) 28/8 (1226-1230).
ISSN: 0008-6363 CODEN: CVREAU
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 24 OF 25 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI The effects of nutrients on lipoprotein susceptibility to oxidation.
ACCESSION NUMBER: 92122606 EMBASE
DOCUMENT NUMBER: 1992122606
TITLE: The effects of nutrients on lipoprotein susceptibility to oxidation.
AUTHOR: Berry E.M.
CORPORATE SOURCE: Israel
SOURCE: Current Opinion in Lipidology, (1992) 3/1 (5-11).
ISSN: 0957-9672 CODEN: COPLEU
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

L6 ANSWER 25 OF 25 DGENE COPYRIGHT 2005 The Thomson Corp on STN
TI Producing a reconstructed oocyte for xenotransplantation purposes comprises whole cell injection of donor cells into an enucleated oocyte to form a reconstructed oocyte.
AN ADM07211 DNA DGENE
AB The invention describes a method of producing a reconstructed oocyte. The method comprises selecting one or more recipient oocytes from a mammal of specific species, enucleating the selected recipient oocytes, selecting one or more somatic donor cells from a donor cell source, injecting a whole cell from the donor cells into an enucleated oocyte to form a reconstructed oocyte, and culturing the reconstructed oocyte under conditions to ensure development of the reconstructed oocyte to a further developmental stage. The methods are useful for producing cloned mammals based on whole cell intracytoplasmic microinjection. The cloned animals may be used as bioreactors to produce proteins of potential value expressed from genes introduced into the cloned animal through genetic engineering techniques. The cloned animal cells or tissues for xenotransplantation may be used to treat diseases, conditions and disabilities including Parkinson's and Alzheimer's diseases, spinal cord injury, stroke, burns, **heart disease**, diabetes, osteoarthritis or rheumatoid arthritis. The significant advantages of the method over the approaches currently available are: the method saves time and labour during the nuclear transfer process essential to successful cloning; the method reduces the extent of oocyte manipulation required in the cloning process; and the method improves the resulting efficiency of cloning. This sequence represents a primer used to detect transgene alphaLA-pLF comprising porcine **lactoferrin** (pLF) driven by the lactoalbumin promoter (alphaLA) to assist detection of cloned piglet embryos.
ACCESSION NUMBER: ADM07211 DNA DGENE
TITLE: Producing a reconstructed oocyte for xenotransplantation purposes comprises whole cell injection of donor cells into an enucleated oocyte to form a reconstructed oocyte.
INVENTOR: Yang X; Lee J
PATENT ASSIGNEE: (YANG-I)YANG X.

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      (LEEJ-I)    LEE J.
PATENT INFO:      US 2004077077 A1 20040422                15p
APPLICATION INFO: US 2002-274432       20021018
PRIORITY INFO:    US 2002-274432       20021018
DOCUMENT TYPE:    Patent
LANGUAGE:         English
OTHER SOURCE:     2004-340006 [31]
DESCRIPTION:      Lactoferrin (pLF)-lactoalbumin promoter (alphaLA)
                  transgene primer #1.

=> d his

     (FILE 'HOME' ENTERED AT 15:53:22 ON 16 MAR 2005)

     FILE 'MEDLINE, JAPIO, BIOSIS, WPIDS, JICST-EPLUS, BIOTECHDS, HCAPLUS,
     SCISEARCH, CEN, CEABA-VTB, BIOBUSINESS, EMBASE, DGENE' ENTERED AT
     15:54:03 ON 16 MAR 2005
L1             1 S LACTOFERRIN AND (REDUCE CIRCULATING LEVELS OF CHOLESTEROL?)
L2             0 S LACTOFERRIN COMPOSITION ADJ2 ADMINISTRATION
L3            18 S LACTOFERRIN COMPOSITION
L4         26082 S LACTOFERRIN
L5             0 S L4 AND (REDUCE VASCULAR INFLAMMATION?)
L6            25 S HEART DISEASE AND L4

=> s l4 and diabetes
L7           205 L4 AND DIABETES

=> s l4 and hypertension
   4 FILES SEARCHED...
L8            51 L4 AND HYPERTENSION

=> s l7 and treatment
L9            38 L7 AND TREATMENT

=> d l8 and treatment
'AND' IS NOT A VALID FORMAT
'TREATMENT' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files.  Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> s l8 and treatment
L10           16 L8 AND TREATMENT

=> d l10 ti abs ibib tot

L10  ANSWER 1 OF 16  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation  on STN
TI   Comparative study of lactoferrin and other blood markers of
     inflammatory stress between preeclamptic and normal pregnancies.
AB   Objective: To test lactoferrin as a blood discriminator of
     neutrophil activation between normal and preeclamptic pregnancy.  Design:
     Comparative study between normal (n = 40) and preeclamptic women receiving
     treatment (n = 42) in the third trimester of pregnancy and in the
     post partum period (30 women with normal pregnancy and 22 with
     preeclampsia).  Methods: Blood, serum or plasma measurements of
     neutrophils, lactoferrin, vitamin C, vitamin E, lipid
     peroxidation products, elastase, C-reactive protein (CRP),
     gamma-glutamyltranspeptidase (gamma-GT), haptoglobin, osmotic fragility,
     urea, creatinine, uric acid, transaminases (ASAT, ALAT), lactic
     dehydrogenase (LDH), platelets, red and white blood cells.  Results: In
     preeclamptic women the ratios of lactoferrin per neutrophil or
     per erythrocyte are higher before delivery than in normal women but
     decrease after delivery.  Delivery induces a greater inflammatory response
     in normal pregnancy as detected by blood concentrations of inflammatory
     markers and hepatic and renal parameters.  Conclusion: Whereas in normal
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pregnant women neutrophil activation increases with delivery, in preeclamptic women the opposite occurs.

ACCESSION NUMBER: 1996:227349 BIOSIS
DOCUMENT NUMBER: PREV199698783478
TITLE: Comparative study of **lactoferrin** and other blood markers of inflammatory stress between preeclamptic and normal pregnancies.
AUTHOR(S): Rebelo, Irene [Reprint author]; Carvalho-Guerra, F.; Perira-Leite, L.; Quintanilha, Alexandre
CORPORATE SOURCE: Dep. Bioquimica, Fac. Farm., Univ. Porto, Rua Anibal Cunha 164, 4000 Porto, Portugal
SOURCE: European Journal of Obstetrics and Gynecology and Reproductive Biology, (1996) Vol. 64, No. 2, pp. 167-173. CODEN: EOGRAL. ISSN: 0301-2115.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 8 May 1996
Last Updated on STN: 8 May 1996

L10 ANSWER 2 OF 16 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Composition for supplementing nutritional deficiencies comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc, and copper.
AN 2004-634381 [61] WPIDS
CR 2004-440316 [41]
AB US2004166175 A UPAB: 20040923
NOVELTY - Composition for supplementing nutritional deficiencies (I) comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc and copper.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a composition (II) comprising (mg) calcium (less than 160, preferably 90 - 110 mg), iron (more than 20, preferably 58.5 - 71.5) and copper (1.8 - 2.2).

ACTIVITY - Anabolic.

A study was undertaken to evaluate the effectiveness of the composition of the present invention in the **treatment** of patients. The objective of the study is to determine whether oral intake of the composition results in an improvement of the nutritional status of a patient in a physiologically stressful state. A double-blind, placebo controlled study was conducted over a six-month period. A total of 120 subjects (60 pregnant women entering the second trimester of pregnancy and 60 lactating women), aged 20 - 35 years, were chosen for the study. An initial assessment of the nutritional status of each woman was conducted utilizing methods such as the peroxide hemolysis test to assess Vitamin E deficiency, measurement of erythrocyte transketolase activity to determine thiamine levels, determination of erythrocyte glutathione reductase activity to assess riboflavin status, and high performance liquid chromatography to directly measure pyridoxine levels. The 120 subjects were separated into four separate groups of 30 women. In a first group comprising only pregnant women and in a second group comprising only lactating women, each subject was administered 2 caplets, daily, of (A1). In a third group comprising only pregnant women and in a fourth group comprising only lactating women, each subject was administered 2 placebo caplets, daily. No other nutritional supplements were taken by the subjects during the assessment period. An assessment of the nutritional status of each woman was conducted utilizing methods such as the peroxide hemolysis test to assess vitamin E deficiency, measurement of erythrocyte transketolase activity to determine thiamine levels, determination of erythrocyte glutathione reductase activity to assess riboflavin status, and high performance liquid chromatography to directly measure pyridoxine levels at one month intervals for a six month period. The data was evaluated using multiple linear regression analysis and a standard t-test. A statistically significant improvement in the nutritional status with respect to vitamin E, thiamine, riboflavin, and pyridoxine was observed in the treated subjects upon completion of the study over the controls.

MECHANISM OF ACTION - None given.

USE - For supplementing nutritional deficiencies to a patient (particularly a pregnant patient or a lactating patient) who is in a

stressful state e.g. a disease state such as pulmonary disorder, a hematological/oncological disorder, a cancer, a disorder of the immune system, a cardiovascular disorder, a hepatic/biliary disorder, a disorder associated with pregnant females and a disorder associated with a fetus. The nutritional deficiencies are a result of elevated metabolic demand, increased plasma volume, decreased concentrations of nutrient-binding proteins (e.g. serum-ferritin, maltose-binding protein, **lactoferrin**, calmodulin, tocopheryl binding protein, riboflavin binding protein, retinol binding protein, transthyretin, high density lipoprotein-apolipoprotein A1, folic acid binding protein, and 25-hydroxyvitamin D binding protein) (all claimed). The disorders associated with pregnant females include osteomalacia and pre-eclampsia and disorders associated with the fetus include neural tube defects and various fetal abnormalities. The pulmonary disorder includes bronchitis, bronchiectasis, atelectasis, pneumonia, diseases caused by inorganic dusts, diseases caused by organic dusts, pulmonary fibrosis and pleurisy. The hematological/oncological disorder includes anemia, hemophilia, leukemia and lymphoma. The disorder of the immune system includes AIDS, AIDS-related complex and bacterial infection. The cardiovascular disorder includes arterial **hypertension**, orthostatic hypotension, arteriosclerosis, coronary artery disease, cardiomyopathy, arrhythmia, valvular heart disease, endocarditis, pericardial disease, cardiac tumor, aneurysm and peripheral vascular disorder. The hepatic/biliary disorder includes jaundice, hepatic steatosis, fibrosis, cirrhosis, hepatitis, hepatic granuloma, liver tumor, cholelithiasis, cholecystitis and choledocholithiasis.

ADVANTAGE - The compositions optimize good health, provide protection against poor nutrition and disease, provide specific nutrients before, during, and after the physiological processes of pregnancy or lactation, which has a profound, positive and comprehensive impact upon the overall wellness of the developing and newborn child as well as the safety and health of the mother.

Dwg.0/0

ACCESSION NUMBER: 2004-634381 [61] WPIDS
CROSS REFERENCE: 2004-440316 [41]
DOC. NO. CPI: C2004-227792
TITLE: Composition for supplementing nutritional deficiencies comprises vitamin A, vitamin D, vitamin C, vitamin E, B-complex, calcium, iron, magnesium, zinc, and copper.
DERWENT CLASS: B04 B05 D13
INVENTOR(S): BALZER, C; GIORDANO, J A
PATENT ASSIGNEE(S): (BALZ-I) BALZER C; (GIOR-I) GIORDANO J A
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2004166175	A1	20040826	(200461)*		12

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2004166175	A1 Cont of	US 2002-315159	20021210
		US 2004-790027	20040302

PRIORITY APPLN. INFO: US 2002-315159 20021210; US 2004-790027 20040302

L10 ANSWER 3 OF 16 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Use of phosphodiesterase-IV along with tumor necrosis factor-alpha for **treatment**/prophylaxis of e.g. pulmonary inflammatory disorders, pulmonary **hypertension** and asthma.
AN 2004-594021 [57] WPIDS
AB WO2004067006 A UPAB: 20040907
NOVELTY - **Treatment** or prophylaxis of a phosphodiesterase-IV (PDE-IV) or a tumor necrosis factor- alpha (TNF- alpha) related condition

comprises administration of a PDE IV inhibitor (A) together with a TNF-alpha antagonist (B).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for (1) a composition comprising (A) and a TNF-alpha antagonist (B) and a pharmaceutically acceptable excipient; and

(2) a kit for the **treatment** or prophylaxis of a PDE IV or a TNF-alpha related condition comprising a dosage form comprising (A) and a dosage form comprising (B).

ACTIVITY - Antiinflammatory; Respiratory-Gen.; Hypotensive; Antiasthmatic; Antiallergic; Antiarthritic; Osteopathic; Ophthalmological; Antidiabetic; Antiangiogenic; Antirheumatic; Neuroprotective.

MECHANISM OF ACTION - Phosphodiesterase-IV (PDE IV) inhibitor; TNF-alpha antagonist. Test details are described for TNF- alpha antagonistic activity but no results given.

USE - (A) along with (B) is useful in the **treatment** of PDE-IV or TNF- alpha related conditions (claimed) such as inflammatory disorders e.g. pulmonary inflammatory disorders, pulmonary **hypertension**, asthma, exercise induced asthma, pollution induced asthma, allergy induced asthma, chronic obstructive pulmonary disorder (COPD), osteoarthritis, adult respiratory distress syndrome, infant respiratory distress syndrome, retinitis, uveitis, glaucoma, retinopathy, diabetic angiopathy, edema formation, arthritis, rheumatoid arthritis and multiple sclerosis.

Dwg.0/0

ACCESSION NUMBER: 2004-594021 [57] WPIDS
DOC. NO. CPI: C2004-216075
TITLE: Use of phosphodiesterase-IV along with tumor necrosis factor-alpha for **treatment**/prophylaxis of e.g. pulmonary inflammatory disorders, pulmonary **hypertension** and asthma.
DERWENT CLASS: B02 B03
INVENTOR(S): WARNER, J M
PATENT ASSIGNEE(S): (PHAA) PHARMACIA CORP
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004067006	A1	20040812	(200457)*	EN	66

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004067006	A1	WO 2004-IB616	20040123

PRIORITY APPLN. INFO: US 2003-442881P 20030127

L10 ANSWER 4 OF 16 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Ophthalmic solution useful for the **treatment** of increased intraocular pressure comprises a prostaglandin of the F-series and an antimicrobial peptide.
AN 2004-011506 [01] WPIDS
AB WO2003079997 A UPAB: 20040102
NOVELTY - An ophthalmic solution comprises a prostaglandin of the F-series and an antimicrobial peptide.

ACTIVITY - Hypotensive; Ophthalmological.
No biological data given.
MECHANISM OF ACTION - None given.
USE - For the **treatment** of increased intraocular pressure,

such as caused by glaucoma and for the reduction of ocular **hypertension**.

ADVANTAGE - The prostaglandin and the antimicrobial peptide work synergistically, to provide beneficial reduction in the incidence of irritant and toxic side effects such as hyperemia, irritation and inflammation of conjunctiva, ocular cell dysplasia, iridial melanocyte hyperplasia, and hyperpigmentation, associated with the prior art prostaglandin compositions. The composition does not contain systemic chemical preservatives such as benzalkonium chloride, BHT or similar irritating phenyl-aromatic preservatives, but instead contains antimicrobial peptides of human eye origin such as **lactoferrin**, thus the composition is suitable for dissension in multidose format, and has improved patient comfort, compliance, acceptance and safety.

Dwg.0/0

ACCESSION NUMBER: 2004-011506 [01] WPIDS
DOC. NO. CPI: C2004-003214
TITLE: Ophthalmic solution useful for the **treatment** of increased intraocular pressure comprises a prostaglandin of the F-series and an antimicrobial peptide.
DERWENT CLASS: B02 B05
INVENTOR(S): JOHNSON, J; MAXEY, K M
PATENT ASSIGNEE(S): (CAYM-N) CAYMAN CHEM CO
COUNTRY COUNT: 103
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003079997	A2	20031002	(200401)*	EN	11
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2003222049	A1	20031008	(200432)		
EP 1501530	A2	20050202	(200510)	EN	
R: AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LU MC NL PT RO SE SI SK TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003079997	A2	WO 2003-US8935	20030321
AU 2003222049	A1	AU 2003-222049	20030321
EP 1501530	A2	EP 2003-718033	20030321
		WO 2003-US8935	20030321

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003222049	A1 Based on	WO 2003079997
EP 1501530	A2 Based on	WO 2003079997

PRIORITY APPLN. INFO: US 2002-367071P 20020321

L10 ANSWER 5 OF 16 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Treating liver dysfunction or parenchyma damage - by admin. of **lactoferrin**, e.g. for improving excretory, detoxification, conjugation and synthesis functions.
AN 1996-078107 [09] WPIDS
AB DE 4426165 A UPAB: 19960305
The use of **lactoferrin**(I) is claimed for the prophylaxis and therapy of liver function disorders and/or liver parenchyma damage and their sequelae. (I) is opt. used in combination with other substances or proteins.

USE - The liver dysfunction treated or prevented specifically results from inflammatory or toxic damage to liver cells, hepatic congestion or liver parenchyma damage. The disorder is specifically excretory, conjugation or synthesis dysfunction, and may be lead to hepatic deficiency coma or be combined with bile duct occlusion. The liver parenchyma damage is specifically toxic damage, fatty degeneration or necrosis of liver cells or liver fibrosis or cirrhosis, and may lead to portal **hypertension**. (I) is especially used for prophylaxis or **treatment** of intoxication by ammonia or protein degradation after liver dysfunction (claimed). (I) may also be used as hepatoprotective agent for improving the detoxification and metabolic function of a transplanted liver.

ADVANTAGE - (I) generally improves liver function; rapidly reduces elevated blood levels of liver enzymes in patients with hepatic insufficiency due to alcohol-induced toxic liver damage; improves protein synthesis, increases blood transferrin, fibrinogen and albumin levels and reduces plasma bilirubin levels in hepatic insufficiency patients; and improves detoxification, e.g. in reducing blood ammonia levels in patients with severe liver dysfunction.

Dwg.0/0

ACCESSION NUMBER: 1996-078107 [09] WPIDS
DOC. NO. CPI: C1996-025888
TITLE: Treating liver dysfunction or parenchyma damage - by admin. of **lactoferrin**, e.g. for improving excretory, detoxification, conjugation and synthesis functions.
DERWENT CLASS: B04
PATENT ASSIGNEE(S): (NITS-N) NITSCHE GMBH H
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
DE 4426165	A1	19960125	(199609)*		7
DE 4426165	C2	19990610	(199927)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
DE 4426165	A1	DE 1994-4426165	19940723
DE 4426165	C2	DE 1994-4426165	19940723

PRIORITY APPLN. INFO: DE 1994-4426165 19940723

L10 ANSWER 6 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Gene expression profiles and biomarkers for the detection of hyperlipidemia and other disease-related gene transcripts in blood
AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular hyperlipidemia, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in **hypertension**, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular **treatment** regimen.
ACCESSION NUMBER: 2005:156681 HCAPLUS
Correction of: 2005:60757
DOCUMENT NUMBER: 142:216629
Correction of: 142:132329

TITLE: Gene expression profiles and biomarkers for the detection of hyperlipidemia and other disease-related gene transcripts in blood
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 39
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004248170	A1	20041209	US 2004-812777	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004248170	A1	20041209	US 2004-812777	20040330
US 2004248170	A1	20041209	US 2004-812777	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812777	A	20040330

L10 ANSWER 7 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Gene expression profiles and biomarkers for the detection of Chagas disease and other disease-related gene transcripts in blood
AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular Chagas disease, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in **hypertension**, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular **treatment** regimen. [This abstract record is one of 3 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].
ACCESSION NUMBER: 2005:60760 HCAPLUS
Correction of: 2004:1036573
DOCUMENT NUMBER: 142:153477

Correction of: 142:16776
TITLE: Gene expression profiles and biomarkers for the detection of Chagas disease and other disease-related gene transcripts in blood
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 154 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 39
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241729	A1	20041202	US 2004-813097	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241729	A1	20041202	US 2004-813097	20040330
US 2004241729	A1	20041202	US 2004-813097	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-813097	A	20040330

L10 ANSWER 8 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Gene expression profiles and biomarkers for the detection of lung disease-related and other disease-related gene transcripts in blood
AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing and monitoring diseases using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in **hypertension**, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention also describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular **treatment** regimen.
ACCESSION NUMBER: 2005:60759 HCAPLUS
Correction of: 2004:1036572
DOCUMENT NUMBER: 142:111840
Correction of: 142:16824
TITLE: Gene expression profiles and biomarkers for the

detection of lung disease-related and other disease-related gene transcripts in blood

INVENTOR(S): Liew, Choong-Chin

PATENT ASSIGNEE(S): Chondrogene Limited, Can.

SOURCE: U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 39

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241728	A1	20041202	US 2004-812764	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241728	A1	20041202	US 2004-812764	20040330
US 2004241728	A1	20041202	US 2004-812764	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812764	A	20040330

L10 ANSWER 9 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN

TI Gene expression profiles and biomarkers for the detection of hyperlipidemia and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular hyperlipidemia, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in **hypertension**, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular **treatment** regimen.

ACCESSION NUMBER: 2005:60757 HCAPLUS
Correction of: 2004:1060658

DOCUMENT NUMBER: 142:132329
Correction of: 142:33757

TITLE: Gene expression profiles and biomarkers for the detection of hyperlipidemia and other disease-related

gene transcripts in blood
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004248170 A1		20041209	US 2004-812777	20040330
PRIORITY APPLN. INFO.:			US 1999-PV115125	19990106
			US 2000-477148	20000104
			US 2002-268730	20021009
			US 2003-601518	20030620
			US 2004-802875	20040312

L10 ANSWER 10 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Analysis of genetic information contained in peripheral blood for diagnosis, prognosis and monitoring **treatment** of allergy, infection and genetic disease in human
AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular allergy, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in **hypertension**, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular **treatment** regimen. [This abstract record is one of 3 recordsfor this document necessitated by thelarge number of index entries required tofully index the document and publicationsystem constraints.].
ACCESSION NUMBER: 2005:60755 HCAPLUS
Correction of: 2004:1036570
DOCUMENT NUMBER: 142:154259
Correction of: 142:36938
TITLE: Analysis of genetic information contained in peripheral blood for diagnosis, prognosis and monitoring **treatment** of allergy, infection and genetic disease in human
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 155 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 39
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004241726	A1	20041202	US 2004-812707	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004241726	A1	20041202	US 2004-812707	20040330
US 2004241726	A1	20041202	US 2004-812707	20040330
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,				

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GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

PRIORITY APPLN. INFO.:

US 1999-115125P	P	19990106
US 2000-477148	B1	20000104
US 2002-268730	A2	20021009
US 2003-601518	A2	20030620
US 2004-802875	A2	20040312
US 2001-271955P	P	20010228
US 2001-275017P	P	20010312
US 2001-305340P	P	20010713
US 2002-85783	A2	20020228
US 2004-809675	A	20040325
US 2004-812707	A	20040330

L10 ANSWER 11 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN

TI Gene expression profiles and biomarkers for the detection of depression-related and other disease-related gene transcripts in blood

AB The present invention is directed to detection and measurement of gene transcripts and their equivalent nucleic acid products in blood. Specifically provided is anal. performed on a drop of blood for detecting, diagnosing, and monitoring diseases, and in particular mental depression, using gene-specific and/or tissue-specific primers. Affymetrix Human Genome U133 and ChondroChip microarrays were used to detect differentially expressed gene transcripts in **hypertension**, obesity, allergy, systemic steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver cancer, schizophrenia, Chagas disease, asthma, and manic depression syndrome. The present invention describes methods by which delineation of the sequence and/or quantitation of the expression levels of disease-specific genes allows for an immediate and accurate diagnostic/prognostic test for disease or to assess the effect of a particular **treatment** regimen.

ACCESSION NUMBER: 2005:1997 HCAPLUS
DOCUMENT NUMBER: 142:111841
TITLE: Gene expression profiles and biomarkers for the detection of depression-related and other disease-related gene transcripts in blood
INVENTOR(S): Liew, Choong-Chin
PATENT ASSIGNEE(S): Chondrogene Limited, Can.
SOURCE: U.S. Pat. Appl. Publ., 154 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 39
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004265868	A1	20041230	US 2004-812702	20040330
US 2004014059	A1	20040122	US 2002-268730	20021009
US 2004248169	A1	20041209	US 2004-812737	20040330
US 2004265869	A1	20041230	US 2004-812716	20040330
US 2004265868	A1	20041230	US 2004-812702	20040330
US 2004265868	A1	20041230	US 2004-812702	20040330
WO 2004112589	A2	20041229	WO 2004-US20836	20040621

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LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,

NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
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AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

PRIORITY APPLN. INFO.:	US 1999-115125P	P	19990106
	US 2000-477148	B1	20000104
	US 2002-268730	A2	20021009
	US 2003-601518	A2	20030620
	US 2004-802875	A2	20040312
	US 2001-271955P	P	20010228
	US 2001-275017P	P	20010312
	US 2001-305340P	P	20010713
	US 2002-85783	A2	20020228
	US 2004-809675	A	20040325
	US 2004-812702	A	20040330

L10 ANSWER 12 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Compositions comprising recombinant **lactoferrin** and its variants in the **treatment** of diabetes mellitus
AB The present invention relates to methods of using a composition of **lactoferrin** for the **treatment** of diabetes mellitus as manifested by a reduction in the levels of serum glucose, blood pressure, obesity, or glycosylated Hb (HbA1c).

ACCESSION NUMBER:	2004:1033558 HCAPLUS
DOCUMENT NUMBER:	141:420455
TITLE:	Compositions comprising recombinant **lactoferrin** and its variants in the **treatment** of diabetes mellitus
INVENTOR(S):	Engelmayer, Jose; Varadhachary, Atul
PATENT ASSIGNEE(S):	Agennix Incorporated, USA
SOURCE:	PCT Int. Appl., 32 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004103285	A2	20041202	WO 2004-US14985	20040513
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2005004006	A1	20050106	US 2004-844865	20040513
PRIORITY APPLN. INFO.:			US 2003-470549P	P 20030514

L10 ANSWER 13 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Prostaglandin F2α analogs in combination with antimicrobial proteins for the **treatment** of glaucoma
AB An ophthalmic formulation for the **treatment** of glaucoma and intraocular pressure comprises a prostaglandin compound of the F-series (PGF), and particularly a prodrug form of a PGF2α analog, such as an ester, amide, or internal lactone, wherein the preservative is an antimicrobial peptide, such as **lactoferrin**. In particularly preferred embodiments, the prostaglandin compound is a macrocyclic internal 1,15-lactone, such as the 16-aryloxy prostaglandin analogs, e.g., fluprostenol or cloprostenol. Thus, a formulation contained fluprostenol

1,15-lactone 0.002, Dextran-70 0.10, HPMC 0.30, NaCl 0.77,KCl 0.12, human recombinant **lactoferrin** 0.10, HCl and/or NaOH to pH 7.0-7.6, and water qs to 100%.

ACCESSION NUMBER: 2003:777542 HCAPLUS
DOCUMENT NUMBER: 139:296971
TITLE: Prostaglandin F2α analogs in combination with antimicrobial proteins for the **treatment** of glaucoma
INVENTOR(S): Maxey, Kirk M.; Johnson, Jennifer
PATENT ASSIGNEE(S): Cayman Chemical Company, USA
SOURCE: PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003079997	A2	20031002	WO 2003-US8935	20030321
WO 2003079997	A3	20040212		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
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EP 1501530	A2	20050202	EP 2003-718033	20030321
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.: US 2002-367071P P 20020321
WO 2003-US8935 W 20030321
OTHER SOURCE(S): MARPAT 139:296971

L10 ANSWER 14 OF 16 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Compositions for improving lipid metabolism
AB A medicinal composition contains as the active ingredient at least one member selected from the group consisting of **lactoferrin** proteins including **lactoferrin** and conalbumin and enzymically digested products of **lactoferrin** proteins including lactoferricin and peptides of conalbumin corresponding to lactoferricin. The composition is useful for improving lipid metabolism For example, it is useful in treating lifestyle-related diseases such as hypercholesterolemia, hypertriglyceridemia, low-d. lipoprotein hypercholesterolemia, high-d. lipoprotein hypocholesterolemia, obesity, fat liver, cholesterol cholelithiasis, severe obesity, hyperlipidemia, **hypertension**, type II diabetes. The composition can elevate basal metabolic rate.

ACCESSION NUMBER: 2003:551399 HCAPLUS
DOCUMENT NUMBER: 139:90498
TITLE: Compositions for improving lipid metabolism
INVENTOR(S): Harada, Etsumori; Takeuchi, Takashi; Ando, Kunio; Shimizu, Hirohiko
PATENT ASSIGNEE(S): Nuclear Receptor Ligand Co., Ltd., Japan
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003057245	A1	20030717	WO 2002-JP13858	20021227
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1466621 A1 20041013 EP 2002-793463 20021227
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

US 2005020484 A1 20050127 US 2004-500245 20040625

PRIORITY APPLN. INFO.: JP 2001-400641 A 20011228
WO 2002-JP13858 W 20021227

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 15 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Biogenic peptides and their potential use.

AB This paper reviews bioactive peptides, biogenic peptides, opioid peptides, immunostimulating peptides, mineral soluble peptides, antihypertensive peptides and antimicrobial peptides originating from food materials and enzymatic hydrolysis of proteins. Antihypertensive peptides are extensively reviewed and have been divided into angiotensin I-converting enzyme inhibitory peptides and others. These peptides are produced in the enzymatic hydrolysate of treated food materials such as milk, animal and fish meat, maize, wheat, soybeans and egg, and also from microbe-fermented products. Peptides with strong antihypertensive effects on spontaneously hypertensive rats are discussed and are divided into high and low angiotensin I-converting enzyme inhibitory activities. In addition, new topics from our studies on antihypertensive peptides are introduced. Efficacies of these peptides in clinical studies and differences with medicinal substances are summarized. Recent studies in this area shown the possibility of using biogenic peptides for improvements in **treatment** or prevention of **hypertension**.

ACCESSION NUMBER: 2003205028 EMBASE
TITLE: Biogenic peptides and their potential use.
AUTHOR: Yamamoto N.; Ejiri M.; Mizuno S.
CORPORATE SOURCE: N. Yamamoto, R/D Center, Calpis Co. Ltd.; 11-10, 5-Chome, Fuchinobe, Sagamihara, Kanagawa 229, Japan. naoyuki.yamamoto@calpis.co.jp
SOURCE: Current Pharmaceutical Design, (2003) 9/16 (1345-1355). Refs: 95
ISSN: 1381-6128 CODEN: CPDEFP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L10 ANSWER 16 OF 16 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI The microcirculation in venous **hypertension**.

AB Objective: To review the factors that result in skin ulceration of patients with chronic venous insufficiency. Data sources: Index Medicus was searched using an on-line computer system for years 1966-1995 to identify articles relating to venous ulceration and the microcirculation. Data extraction: Articles and section of articles relating to the mechanisms which cause venous ulceration and the efficacy of the **treatment** of venous ulceration have been included. Data synthesis: It seems unlikely that venous ulceration is attributable to failure of diffusion of oxygen and other small nutritional molecules to the tissues of the skin. It is much more likely that neutrophils attach themselves to

the cutaneous microcirculation, become activated and produce endothelial injury. Repeated over many months or years, this leads to the chronic inflammatory process of lipodermatosclerosis. The microvascular changes in the skin are characterised by activated endothelium and perivascular inflammatory cells. Conclusion: There is evidence of leucocyte involvement in the pathogenesis of venous ulceration. The exact mechanisms remain to be resolved. Improved **treatment** for patients may be devised with a better understanding of the basic causes of this condition.

ACCESSION NUMBER: 96304872 EMBASE
DOCUMENT NUMBER: 1996304872
TITLE: The microcirculation in venous **hypertension**.
AUTHOR: Coleridge Smith P.D.
CORPORATE SOURCE: Department of Surgery, UCL Medical School, The Middlesex Hospital, Mortimer Street,London W1N 8AA, United Kingdom
SOURCE: Cardiovascular Research, (1996) 32/4 (789-795).
ISSN: 0008-6363 CODEN: CVREAU
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 002 Physiology
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English

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     (FILE 'HOME' ENTERED AT 15:53:22 ON 16 MAR 2005)

     FILE 'MEDLINE, JAPIO, BIOSIS, WPIDS, JICST-EPLUS, BIOTECHDS, HCAPLUS,
     SCISEARCH, CEN, CEABA-VTB, BIOBUSINESS, EMBASE, DGENE' ENTERED AT
     15:54:03 ON 16 MAR 2005
L1             1 S LACTOFERRIN AND (REDUCE CIRCULATING LEVELS OF CHOLESTEROL?)
L2             0 S LACTOFERRIN COMPOSITION ADJ2 ADMINISTRATION
L3            18 S LACTOFERRIN COMPOSITION
L4         26082 S LACTOFERRIN
L5             0 S L4 AND (REDUCE VASCULAR INFLAMMATION?)
L6            25 S HEART DISEASE AND L4
L7           205 S L4 AND DIABETES
L8            51 S L4 AND HYPERTENSION
L9            38 S L7 AND TREATMENT
L10           16 S L8 AND TREATMENT

=> s lactoferrin adj2 cholesterol
L11            0 LACTOFERRIN ADJ2 CHOLESTEROL

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'THERAPY' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
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or the STNGUIDE file for information on formats available in
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L15           33 L14 AND L13

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L16            8 L15 AND L12
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L16 ANSWER 1 OF 8 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Radio-labelling a biomolecule useful for **treatment** of e.g. cancer involves contacting biomolecule with radionuclide in presence of weak transfer ligand.
AN 2003-689623 [65] WPIDS
AB WO2003068270 A UPAB: 20031009
NOVELTY - Radio-labelling a biomolecule involves contacting the biomolecule with radionuclide in the presence of a weak transfer ligand.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for;
(1) a kit comprising a biomolecule, radionuclide and a weak transfer ligand and optionally a set of written instructions;
(2) a radionuclide-labelled product;
(3) a product comprising technetium labelled iron transport protein (preferably **lactoferrin**) coupled to a chemotherapeutic agent;
(4) a composition comprising **lactoferrin**, radiolabelled **lactoferrin** or technetium labelled **lactoferrin** coupled to a chemotherapeutic agent;
(5) diagnosing the presence of a tumor involving administering a technetium labelled **lactoferrin** product and imaging the labelled product in the body; and
(6) **treatment** of tumor involving administering a composition comprising a chemotherapeutic or gene **therapy** agent coupled to technetium labelled transferrin or **lactoferrin**.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - None given.
USE - In the manufacture of a medicament for the **treatment** of cancer and tumor (claimed) e.g. breast cancer, bladder carcinoma, lung and **heart** tumor.
ADVANTAGE - The method removes extraneous radionuclide material, leading to high labelling efficiencies and pure radionuclide-labelled materials and improves purity of radio-labelled.
Dwg.0/11
ACCESSION NUMBER: 2003-689623 [65] WPIDS
DOC. NO. CPI: C2003-189108
TITLE: Radio-labelling a biomolecule useful for **treatment** of e.g. cancer involves contacting biomolecule with radionuclide in presence of weak transfer ligand.
DERWENT CLASS: B04 K08
INVENTOR(S): SMITH, T; WALTON, P
PATENT ASSIGNEE(S): (VIST-N) VISTATEC YORK LTD
COUNTRY COUNT: 103
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003068270	A1	20030821	(200365)*	EN	22
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
GB 2388605	A	20031119	(200401)		
AU 2003245691	A1	20030904	(200428)		
EP 1482988	A1	20041208	(200480)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003068270	A1	WO 2003-GB548	20030207

GB 2388605	A	GB 2003-3005	20030211
AU 2003245691	A1	AU 2003-245691	20030207
EP 1482988	A1	EP 2003-739552	20030207
		WO 2003-GB548	20030207

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003245691	A1 Based on	WO 2003068270
EP 1482988	A1 Based on	WO 2003068270

PRIORITY APPLN. INFO: GB 2002-15511 20020705; GB 2002-3330 20020212

L16 ANSWER 2 OF 8 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI New probiotic nutritional preparation comprising Bifidobacterium, Enterococcus faecium and Lactobacillus is useful for treating gastrointestinal disorders.
AN 1999-192461 [17] WPIDS
AB EP 904784 A UPAB: 20011203
NOVELTY - The nutritional preparation with health promoting action, especially for treating gastrointestinal tract disorders comprises 106-1014(especially 107-1013) viable cells (especially Bifidobacterium, Enterococcus faecium and a Lactobacillus strain that predominantly produces dextro-rotatory lactate) /g of total preparation.

ACTIVITY - Antiinflammatory; antibiotic; antidiarrheic; antifungal; antiviral; antirheumatic.

MECHANISM OF ACTION - Immuno stimulant; **heart** and blood circulation stimulant

USE - The nutritional preparation is useful:

(1) in the form of a food supplement preferably comprising a freeze-dried preparation of the microorganisms; and

(2) in the form of a food composition ready for consumption obtained by either:

(i) mixing the microorganisms with a suitable food or food base;

(ii) cultivating the microorganisms in a suitable food or food base; or

(iii) adding a supplement (as above) to a suitable food or food base (all claimed).

The nutritional preparation is useful in preventing and treating gastrointestinal tract disorders e.g. gastrointestinal infections, diarrhea, systemic infections or disturbances in the immune system especially those caused by pathogens e.g. enterotoxigenic E. coli strains, rotaviruses, Clostridia, Salmonella and/or Campylobacter sp. and further for the **therapy** and prophylaxis of IBS, Crohn's disease, cancer of the GI tract, impaired immune function against bacteria, fungi (e.g. Candida albicans), yeasts and/or viruses, obstipation, antibiotics **therapy** or radiotherapy and/or disorders of the **heart** or blood circulation, acute rheumatics and/or vaginitis. No specific examples given.

ADVANTAGE - Encapsulation of the microorganisms enable improved shelf-life of over two years especially to food products which are ready for consumption and are entirely safe as a food supplement. Encapsulation can also improve further the resistance against stomach acids and/or pancreatic fluid. The nutritional preparation prevents colonisation of harmful organisms after the **treatment** has ended or for restoring the gastrointestinal flora after antibiotic **treatment** and comprises different microorganisms that colonise and grow in different parts of the gastrointestinal tract therefore providing overall **treatment**.

Dwg.0/0

ACCESSION NUMBER: 1999-192461 [17] WPIDS
DOC. NO. CPI: C1999-056688
TITLE: New probiotic nutritional preparation comprising Bifidobacterium, Enterococcus faecium and Lactobacillus is useful for treating gastrointestinal disorders.
DERWENT CLASS: B04 D13 D16

INVENTOR(S): HAGEMAN, R J J; VAN HOEY-DE-BOER, K A
PATENT ASSIGNEE(S): (NUTR-N) NUTRICIA NV
COUNTRY COUNT: 18
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 904784	A1	19990331	(199917)*	EN	9

R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 904784	A1	EP 1997-202900	19970922

PRIORITY APPLN. INFO: EP 1997-202900 19970922

L16 ANSWER 3 OF 8 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Use of human apo-**lactoferrin** and peptides derivable from human **lactoferrin** for the production of composition useful for e.g. treating and preventing vascular disease;
human apo-**lactoferrin** for use in disease **therapy**
AN 2003-25156 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - In the production of a composition, a substance containing human apo-**lactoferrin** and/or peptides derivable from human **lactoferrin** and/or its natural metabolites or equivalent analogs is used.
ACTIVITY - Antianginal; Cerebroprotective; Cardiant; Antiulcer; Antialopecia.
MECHANISM OF ACTION - VEGF165 induced angiogenesis inhibitor. **Lactoferrin**, dissolved in saline, was given by tube feeding twice daily from Sunday afternoon (Day-1) to Friday afternoon (Day 4). Vehicle controls received saline by tube feeding. The angiogenesis **treatment** with VEGF was given intraperitoneally on Days 0 - 4 (twice daily). The results for test/control groups were vascularized area = 12.09+/- 1.49/1.18+/- 0.5, microvascular length = 1.465+/- 0.077/0.28+/- 0.04, and total microvascular length = 17.72+/- 2.19/0.33+/- 0.14 respectively. The results demonstrated that oral administration of apo-hLE significantly enhanced the VEGF mediated angiogenic response.
USE - For treating and/or preventing vascular disease and/or states of tissue hypoperfusion (including impending or manifested stroke, ischemic **heart** disease e.g. angina pectoris or impending or manifested myocardial infarction), or peripheral artery occlusive disease with or without impending gangrene and/or state of depressed VEGF induced angiogenesis associated with peptic ulcer, leg ulcer or local or generalized hair loss) with hypoxia and/or ischemic consequences (claimed).
ADMINISTRATION - The route of administration is oral, parenteral, local or by inhalation. No dosage given.
ADVANTAGE - The method is used in as an alternative to bypass surgery or any therapeutic angiogenesis options.
EXAMPLE - No relevant example given. (14 pages)
ACCESSION NUMBER: 2003-25156 BIOTECHDS
TITLE: Use of human apo-**lactoferrin** and peptides derivable from human **lactoferrin** for the production of composition useful for e.g. treating and preventing vascular disease;
human apo-**lactoferrin** for use in disease **therapy**
AUTHOR: NORRBY K
PATENT ASSIGNEE: NORRBY K
PATENT INFO: WO 2003072129 4 Sep 2003
APPLICATION INFO: WO 2003-SE329 27 Feb 2003
PRIORITY INFO: SE 2002-598 27 Feb 2002; SE 2002-598 27 Feb 2002

DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-712670 [67]

L16 ANSWER 4 OF 8 BIOTECHDS COPYRIGHT 2005 THE THOMSON CORP. on STN
TI Radio-labelling a biomolecule useful for **treatment** of e.g. cancer involves contacting biomolecule with radionuclide in presence of weak transfer ligand;
radionuclide label for chemotherapy or gene **therapy**
AN 2003-24609 BIOTECHDS
AB DERWENT ABSTRACT:
NOVELTY - Radio-labelling a biomolecule involves contacting the biomolecule with radionuclide in the presence of a weak transfer ligand.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for; (1) a kit comprising a biomolecule, radionuclide and a weak transfer ligand and optionally a set of written instructions; (2) a radionuclide-labelled product; (3) a product comprising technetium labelled iron transport protein (preferably **lactoferrin**) coupled to a chemotherapeutic agent; (4) a composition comprising **lactoferrin**, radiolabelled **lactoferrin** or technetium labelled **lactoferrin** coupled to a chemotherapeutic agent; (5) diagnosing the presence of a tumor involving administering a technetium labelled **lactoferrin** product and imaging the labelled product in the body; and (6) **treatment** of tumor involving administering a composition comprising a chemotherapeutic or gene **therapy** agent coupled to technetium labelled transferrin or **lactoferrin**.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - None given.
USE - In the manufacture of a medicament for the **treatment** of cancer and tumor (claimed) e.g. breast cancer, bladder carcinoma, lung and **heart** tumor.
ADMINISTRATION - The composition is administered as a single dose or repeatedly by intravenous, intramuscular, subcutaneous or oral route or is injected directly to the tumor site (claimed).
ADVANTAGE - The method removes extraneous radionuclide material, leading to high labelling efficiencies and pure radionuclide-labelled materials and improves purity of radio-labelled.
EXAMPLE - A mixture of 2.8x10-7 mol dm-3 solution of thiourea (25 microl), 10-10 mol dm1-3 solution of SnCl2 (25 microl), pertechnetate (50 microl) obtained from (99m)Tc generator and transferin (25 microl) was taken in a glass vial. The mixture was incubated for 1 hour at 37degreesC followed by addition of phosphate buffered saline (0.85 microl). The solution was further maintained at 37 degrees C for 15 minutes to obtained a radiolabelled biomolecule. (22 pages)
ACCESSION NUMBER: 2003-24609 BIOTECHDS
TITLE: Radio-labelling a biomolecule useful for **treatment** of e.g. cancer involves contacting biomolecule with radionuclide in presence of weak transfer ligand;
radionuclide label for chemotherapy or gene **therapy**
AUTHOR: WALTON P; SMITH T
PATENT ASSIGNEE: VISTATEC YORK LTD
PATENT INFO: WO 2003068270 21 Aug 2003
APPLICATION INFO: WO 2003-GB548 7 Feb 2003
PRIORITY INFO: GB 2002-15511 5 Jul 2002; GB 2002-3330 12 Feb 2002
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-689623 [65]

L16 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
TI Nucleic acid compositions, kits, and methods for identification, assessment, prevention, and **therapy** of human breast cancer
AB The invention relates to nucleic acid marker compns., kits and methods for detecting, characterizing, preventing, and treating human breast cancers. A variety of markers are provided, wherein changes in the levels of expression of one or more of the nucleic acid markers is correlated with the presence of breast cancer. The level of expression of numerous potential markers was measured in cells obtained from breast cancer tissue

samples obtained form fifteen patients afflicted with breast cancer and from eleven breast cancer cell cultures, based on comparison with expression levels of each marker in corresponding non-cancerous breast tissue and cell cultures. The 15 cancer tissue samples include (i) five invasive lobular carcinomas (ILC), (ii) five invasive ductal carcinomas (IDC), and (iii) five samples of ductal carcinoma in situ (DCIS). As an addnl. evaluation of ability to indicate breast cancer, individual markers that were identified by transcriptional profiling criteria were also tested in six different subtracted library expts. In addition, protein profiling expts. were undertaken to assess whether the proteins associated with the expression of individual markers of the invention are secreted. Table 21 lists approx. 43,500 GenBank Accession Nos. from the present invention. [This abstract record is one of 8 records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

ACCESSION NUMBER: 2001:863850 HCAPLUS
DOCUMENT NUMBER: 136:32755
TITLE: Nucleic acid compositions, kits, and methods for identification, assessment, prevention, and **therapy** of human breast cancer
INVENTOR(S): Lillie, James; Palermo, Adam; Wang, Youzhen; Steinmann, Kathleen; Elias, Josh
PATENT ASSIGNEE(S): Millennium Predictive Medicine, Inc., USA
SOURCE: PCT Int. Appl., 2674 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001046697 A2		20010628	WO 2000-US35214	20001221

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR

PRIORITY APPLN. INFO.:	
US 1999-PV171406	19991221
US 2000-PV176423	20000114
US 2000-PV190471	20000317
US 2000-PV193482	20000329
US 2000-PV205231	20000515
US 2000-PV213236	20000620
US 2000-PV219865	20000720

L16 ANSWER 6 OF 8 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

TI Idiopathic pulmonary hemosiderosis in adults.

AB Idiopathic pulmonary hemosiderosis is a rare interstitial lung disease of unknown etiology, characterized by recurrent episodes of diffuse alveolar hemorrhage. It is most often seen in children, but an adult type has also been described. A constellation of cough, hemoptysis, dyspnea, pulmonary infiltrates, and anemia suggests a diagnosis of diffuse alveolar hemorrhage. Sputum and bronchoalveolar lavage examination show numerous hemosiderin-laden macrophages (siderophages), while the lung biopsy confirms a bland alveolar hemorrhage, variable degrees of interstitial fibrosis, and intraalveolar/interstitial siderophages. The **treatment** of choice consists of immunosuppressive agents, including corticosteroids, for both acute exacerbations and remission periods. With the advent of more effective immunosuppressive agents, long-term disease-free periods and better survival have been described. Terminology: Hemosiderin (Gr. hemo blood + Gr. sideros iron)-an intracellular storage form of iron, found as golden-yellow or brown-yellow granules containing a complex of ferric hydroxides, polysaccharides, and proteins. It contains >33% iron by weight and stains blue with Perls'

Prussian stain. Hemosiderosis-a focal or general increase in tissue iron stores, especially in local macrophages. Pulmonary Hemosiderosis - the deposition of abnormal amounts of hemosiderin in the lungs, especially in the alveolar macrophages and interstitium, due to recurrent alveolar hemorrhage episodes. Can be due to various conditions (congestive **heart** failure, valvulopathies, vasculitides, etc). Idiopathic Pulmonary Hemosiderosis (Heiner-Ceelen disease) -repeated, sudden attacks of dyspnea, hemoptysis, diffuse alveolar bleeding, and anemia, seen mostly in children but also in adults. Hemochromatosis (Gr. hemo + Gr. chromatosis, staining, discoloration)-a disorder due to deposition of hemosiderin in the parenchymal cells, causing tissue damage of the liver, pancreas, **heart**, and pituitary. Siderosis (Gr. sideros + Gr. -osis, condition)-(1) pneumoconiosis siderotica, a form of pneumoconiosis due to the presence of iron dust, usually serious when combined with silica exposure (silicosiderosis); (2) hemosiderosis; (3) hyperferremia. Siderophage (Gr. sideros + Gr. phagos, ingestion)-a macrophage laden with phagocytosed iron-containing particles. Siderophillin-nonheme iron-binding proteins: transferrin (in vertebrate blood), **lactoferrin** (in mammalian milk and other secretions), conalbumin, and ovotransferrin (in avian blood and egg white).

ACCESSION NUMBER: 2005043129 EMBASE
TITLE: Idiopathic pulmonary hemosiderosis in adults.
AUTHOR: Ioachimescu O.C.; Kotch A.; Stoller J.K.
CORPORATE SOURCE: Dr. O.C. Ioachimescu, 9500 Euclid Ave., Cleveland, OH 44195, United States. oioac@yahoo.com
SOURCE: Clinical Pulmonary Medicine, (2005) 12/1 (16-25).
Refs: 116
ISSN: 1068-0640 CODEN: CPMEF2
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

L16 ANSWER 7 OF 8 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Diagnostic and therapeutic strategies in the irritable bowel syndrome.
AB The management of patients with irritable bowel syndrome (IBS) is a frequent, yet challenging task in both primary care and gastroenterology practice. A diagnostic strategy guided by keen clinical judgment should focus on positive symptom criteria and on the absence of alarm symptoms. In younger patients lacking alarm features, invasive testing has a low-yield. The presence of food intolerance and underlying celiac disease should be excluded. The usefulness of fecal tests such as calprotectin and **lactoferrin** to exclude organic bowel disease is not adequately established. In patients with moderate to severe symptoms who fail initial therapeutic trials, further tests can be performed in tertiary care settings, such as transit measurement and tests for diagnosing pelvic floor dysfunction. **Treatment** strategies for IBS are currently directed at the predominant symptoms. In diarrhea-predominant IBS, opioids (e.g. loperamide) and the 5-HT(3) receptor antagonist alosetron are efficacious. In constipation-predominant IBS, fiber and bulk laxatives are traditionally used, but their efficacy is variable and may worsen symptoms. The 5-HT(4) receptor agonist tegaserod is efficacious in female patients with IBS and constipation. In patients with IBS and abdominal pain, antispasmodics and antidepressants can be used, with the best evidence supporting the prescription of tricyclic antidepressants. The efficacy of psychological treatments in terms of relieving the symptoms of IBS is still uncertain. Limited evidence suggests that anti-enkephalinase agents, somatostatin analogues, α(2)-receptor agonists, opioid antagonists, selective serotonin reuptake inhibitors, probiotics and herbal treatments may be useful in IBS patients.

ACCESSION NUMBER: 2004483915 EMBASE
TITLE: Diagnostic and therapeutic strategies in the irritable bowel syndrome.
AUTHOR: Cremonini F.; Talley N.J.
CORPORATE SOURCE: Dr. N.J. Talley, Department of Medicine, Mayo Clinic

College of Medicine, 200 First Street S.W., Rochester, MN 55905, United States. Talley.Nicholas@mayo.edu
SOURCE: Minerva Medica, (2004) 95/5 (427-441).
Refs: 128
ISSN: 0026-4806 CODEN: MIMEAO
COUNTRY: Italy
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 037 Drug Literature Index
038 Adverse Reactions Titles
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English; Italian

L16 ANSWER 8 OF 8 EMBASE
on STN
TI Heparin-coated cardiopulmonary bypass equipment. I. Biocompatibility markers and development of complications in a high-risk population.
AB Objectives: 1. To study possible clinical benefits of heparin-coated cardiopulmonary bypass in patients with a broad range of preoperative risk factors. 2. To evaluate the correlation between the terminal complement complex and clinical outcome. 3. To identify clinical predictors of complement activation and correlates of granulocyte activation during cardiac surgery. Methods: Blood samples from adults undergoing elective cardiac surgery with Duraflo II heparin-coated (n = 81) or uncoated (n = 75) cardiopulmonary bypass sets (Duraflo coating surface; Baxter International, Inc, Deerfield, III) were analyzed for activation of complement (C3 activation products, terminal complement complex), granulocytes (myeloperoxidase, **lactoferrin**), and platelets (β-thromboglobulin) by enzyme immunoassays. Preoperative risk was assessed by means of the 'Higgins' score.' Complications (cardiac, renal, pulmonary, gastrointestinal, and central nervous system dysfunction, infections, death) were registered prospectively. Data were analyzed by analysis of variance, logistic regression, and linear regression. Results and conclusions: Sixty-seven percent of the patients had predefined risk factors. Complications developed in 53 patients (34%), equivalently with and without heparin-coated bypass sets (P = .44-.82), despite a significant reduction in complement and granulocyte activation by heparin coating. No clear-cut relationship between the terminal complement complex and outcome was found, even if it was significant in the models for renal and central nervous system dysfunction and infections (P = .006). The Higgins' score was significantly related to complement activation (P < .05). Approximately 50% of the variation in granulocyte activation was explained by complement (P ≤ .01) and platelet activation (P < .05), heparin/protamine dose ratio (P = .02), duration of cardiopulmonary bypass (P < .01), and gender (P < .05). Therefore measures reducing complement activation alone will not necessarily reduce granulocyte activation sufficiently for clinical significance.
ACCESSION NUMBER: 1999125381 EMBASE
TITLE: Heparin-coated cardiopulmonary bypass equipment. I. Biocompatibility markers and development of complications in a high-risk population.
AUTHOR: Videm V.; Mollnes T.E.; Fosse E.; Mohr B.; Bergh K.; Hagve T.-A.; Aasen A.O.; Svennevig J.L.
CORPORATE SOURCE: Dr. V. Videm, Department of Immunology, Blood Bank, Regional Hospital, N-7006 Trondheim, Norway
SOURCE: Journal of Thoracic and Cardiovascular Surgery, (1999) 117/4 (794-802).
Refs: 28
ISSN: 0022-5223 CODEN: JTCSAQ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
018 Cardiovascular Diseases and Cardiovascular Surgery
027 Biophysics, Bioengineering and Medical Instrumentation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

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E1            1     VARADHACHARY S N/AU
E2            9     VARADHACHARY SEEVARAM N/AU
E3            0 --> VARADHACHARY, A/AU
E4            3     VARADHAN C/AU
E5            1     VARADHAN G/AU
E6            5     VARADHAN J/AU
E7           11     VARADHAN K/AU
E8            1     VARADHAN L/AU
E9           18     VARADHAN R/AU
E10           1     VARADHAN R I/AU
E11          11     VARADHAN R S/AU
E12           9     VARADHAN RAVI/AU

=> e engelmayer, j/au
E1            2     ENGELMAYER ULF/AU
E2            1     ENGELMAYER W/AU
E3            0 --> ENGELMAYER, J/AU
E4            3     ENGELMAYR G C/AU
E5            3     ENGELMAYR GEORGE C/AU
E6            4     ENGELMAYR GEORGE C JR/AU
E7            4     ENGELMAYR J/AU
E8            2     ENGELMAYR JR G C/AU
E9            1     ENGELMEIER/AU
E10          11     ENGELMEIER D/AU
E11           6     ENGELMEIER DORIS/AU
E12           1     ENGELMEIER E/AU

=> e glynn,P/au
E1            1     GLYNN WILLIAM K/AU
E2            1     GLYNN WILLIAMS B/AU
E3            0 --> GLYNN,P/AU
E4            6     GLYNNBARNHART A/AU
E5            3     GLYNNBARNHART A M/AU
E6           36     GLYNNE A/AU
E7            3     GLYNNE ALAN/AU
E8            2     GLYNNE G L/AU
E9            1     GLYNNE GUT I/AU
E10           1     GLYNNE JONES DENYS/AU
E11          38     GLYNNE JONES E/AU
E12           8     GLYNNE JONES EVE/AU

=> e wang, y/au
E1           10     WANG ZY/AU
E2            2     WANG ZYX/AU
E3            0 --> WANG, Y/AU
E4            1     WANG1 Y/AU
E5            1     WANGA A P/AU
E6            1     WANGA C/AU
E7            1     WANGA C C/AU
E8            1     WANGA D/AU
E9            1     WANGA D B/AU
E10           1     WANGA G/AU
E11           1     WANGA G J/AU
E12           1     WANGA GE/AU

=> s LDL decrease and l4
L17           0 LDL DECREASE AND L4

=> s decrease triglyceride and l4
L18           0 DECREASE TRIGLYCERIDE AND L4

=> s decrease VLDL and l4
L19           0 DECREASE VLDL AND L4

=> s (C-reactive protein) and l4
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L20 210 (C-REACTIVE PROTEIN) AND L4

=> d l20 ti abs ibib 1-15

L20 ANSWER 1 OF 210 MEDLINE on STN

TI Early release of neutrophil markers of activation after direct stenting in patients with unstable angina.

AB OBJECTIVE: To assess polymorphonuclear neutrophils activation after stenting in acute coronary syndromes studied by myeloperoxydase, **lactoferrin** and elastase release in this clinical setting. METHODS: Myeloperoxydase, **lactoferrin**, elastase, **C-reactive protein** and cytokines serum levels were assessed in 20 patients undergoing catheterization for unstable angina. Serial sampling starting before arteriography and continued up to 24 h was carried out in 15 patients undergoing direct stenting (group A) and in five patients assessed by coronary angiography only (group B). RESULTS: Myeloperoxydase, **lactoferrin** and elastase levels remained unchanged following catheterization, whereas a significant increase in myeloperoxydase (P=0.0009) and **lactoferrin** (P=0.004) was observed after stenting. No change in levels of tumour necrosis factor alpha, interleukin (IL)-8 and IL-11 was found in group B after catheterization at the different sampling times, although IL-8 and IL-11 levels increased transiently following stenting. IL-6 values increased in both groups. Baseline values of **C-reactive protein** were similar in each group. A progressive increase in **C-reactive protein** was noted in both groups and appeared to be larger following stenting (group A: P=0.0002; group B: P=0.01). CONCLUSIONS: In patients with unstable angina, stenting is associated by immediate neutrophil activation followed by release of inflammatory cytokines (IL-6, IL-8, IL-11) and **C-reactive protein** elevation. This study points out a potential role of myeloperoxydase as a trigger for inflammatory reaction in patients with unstable coronary syndromes undergoing percutaneous coronary intervention.

ACCESSION NUMBER: 2005026124 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15654202
TITLE: Early release of neutrophil markers of activation after direct stenting in patients with unstable angina.
AUTHOR: Gach Olivier; Biemar Christian; Nys Monique; Deby-Dupont Ginette; Chapelle Jean-Paul; Deby Carol; Lamy Maurice; Pierard Luc A; Legrand Victor
CORPORATE SOURCE: Centre Hospitalier Universitaire du Sart-Tilman, Service de Cardiologie, Liege, Belgium.
SOURCE: Coronary artery disease, (2005 Feb) 16 (1) 59-65. Journal code: 9011445. ISSN: 0954-6928.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-DATA-REVIEW; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20050119
Last Updated on STN: 20050119

L20 ANSWER 2 OF 210 MEDLINE on STN

TI The inflammatory response in mild and in severe psoriasis.

AB BACKGROUND: Psoriasis is a chronic and recurrent inflammatory skin disease. The inflammatory response represents a fundamental ability of the organism to protect itself from infectious agents and from injury. OBJECTIVES: To evaluate the inflammatory response in mild and in severe psoriasis, to evaluate the endogenous systems counterbalancing the deleterious effects of the inflammation products, and to establish values of prognostic significance. METHODS: The study was performed in a control group (n = 40) and in 60 patients with psoriasis vulgaris, half presenting with mild psoriasis, and the other half with severe psoriasis. We evaluated total and differential leucocyte count; elastase, **lactoferrin** and lipid peroxidation as markers of neutrophil activation; total plasma antioxidant capacity (TAS), transferrin, ceruloplasmin, alpha(1)-antitrypsin and alpha(2)-macroglobulin as markers

of the endogenous antioxidant and antiprotease systems; and fibrinogen, erythrocyte sedimentation rate, **C-reactive protein** (CRP), haptoglobin, C3 and C4 complement proteins as markers of inflammation. RESULTS: Our data suggested that psoriasis is an inflammatory condition in which neutrophils seem to play a crucial role by contributing to the development of oxidative and proteolytic stress. The worsening of the disease seemed to be linked to the enhancement of the inflammatory response and of the imbalance between neutrophil activation products and their inhibitors. CONCLUSIONS: We propose values for elastase, CRP, elastase/alpha(2)-macroglobulin, elastase/alpha(1)-antitrypsin, thiobarbituric acid/TAS and elastase/neutrophil ratios with prognostic significance for the worsening of psoriasis.

ACCESSION NUMBER: 2004251129 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15149504
TITLE: The inflammatory response in mild and in severe psoriasis.
AUTHOR: Rocha-Pereira P; Santos-Silva A; Rebelo I; Figueiredo A; Quintanilha A; Teixeira F
CORPORATE SOURCE: Departamento de Quimica da Universidade da Beira Interior, Rua Marques d'Avila e Bolama, 6200 Covilha, Portugal.. petrorp@ciunix.ubi.pt
SOURCE: British journal of dermatology, (2004 May) 150 (5) 917-28. Journal code: 0004041. ISSN: 0007-0963.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 20040520
Last Updated on STN: 20040716
Entered Medline: 20040715

L20 ANSWER 3 OF 210 MEDLINE on STN
TI Azurocidin-specific-ANCA-related idiopathic necrotizing crescentic glomerulonephritis.
AB An 80-year-old woman who had rapidly progressive glomerulonephritis unaccompanied by systemic vasculitis is described. On renal biopsy, she showed necrotizing crescentic glomerulonephritis by light microscopy and pauci-immune glomerular lesions by immunofluorescent study. No dense deposits were present on electronmicroscopic study. On serum examination, indirect immunofluorescent study showed perinuclear pattern antineutrophil cytoplasmic antibody (ANCA), but myeloperoxidase-ANCA and proteinase 3-ANCA were both negative. Her serum reacted only to azurocidin excluding other ANCA antigens: bactericidal permeability-increasing protein, cathepsin G, elastase, **lactoferrin**, or lysozyme. Serum creatinine level decreased, and **C-reactive protein** turned negative after steroid therapy. Azurocidin-ANCA also turned negative. It is suggested that azurocidin-ANCA might have been related to the inflammatory process of pauci-immune necrotizing crescentic glomerulonephritis in this patient.

ACCESSION NUMBER: 2004147761 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15042565
TITLE: Azurocidin-specific-ANCA-related idiopathic necrotizing crescentic glomerulonephritis.
AUTHOR: Kimura Rio; Matsuzawa Naoki; Arimura Yoshihiro; Soejima Akinori; Nakabayashi Kimimasa; Yamada Akira
CORPORATE SOURCE: First Department of Internal Medicine, Kyorin University School of Medicine, Tokyo, Japan.. rio@ac.catv.ne.jp
SOURCE: American journal of kidney diseases : official journal of the National Kidney Foundation, (2004 Apr) 43 (4) e7-10. Journal code: 8110075. ISSN: 1523-6838.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 20040326
Last Updated on STN: 20040617

Entered Medline: 20040616

L20 ANSWER 4 OF 210 MEDLINE on STN
TI **C-reactive protein** and antibacterial activity in blood plasma of colostrum-fed calves and the effect of lactulose.
AB Several milk proteins are very important for immunological defense and can be absorbed in the intestine of calves in the first hours after birth. The influence of colostrum intake and the effect of additional lactulose application on the concentration of **C-reactive protein** (CRP) in blood were investigated. The CRP is known as a mediator of innate immunity. Results were compared to the bovine acute phase protein haptoglobin, and to lactalbumin, **lactoferrin**, and immunoglobulins in plasma from calves. After colostrum intake, the concentration of most proteins were strongly increased. The data show, for the first time, a significant increase of CRP in the blood of calves 1 d after colostrum intake (nonlactulose group, n = 10), and an even more significant increase in CRP concentration (1 d postpartum) was measured in the group of animals with additional application of lactulose (lactulose group, n = 10) when compared to the nonlactulose group. In an in vitro assay with the plasma of these animals, an increased bactericidal activity was detected against Morganella morganii (1 d postpartum) in both groups, but again a higher activity occurred in the lactulose group. The results of these investigations emphasize the importance of colostrum intake during the first hours after birth for the defense potential of newborn calves. In addition, lactulose may have a positive effect in the period of passive transfer of colostrum proteins and in the immune defense.
ACCESSION NUMBER: 2003515885 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14594250
TITLE: **C-reactive protein** and antibacterial activity in blood plasma of colostrum-fed calves and the effect of lactulose.
AUTHOR: Schroedl W; Jaekel L; Krueger M
CORPORATE SOURCE: Institute of Bacteriology and Mycology, Veterinary Faculty, University of Leipzig, Leipzig, Germany 04103.. schroedl@rz-uni-leipzig.de
SOURCE: Journal of dairy science, (2003 Oct) 86 (10) 3313-20. Journal code: 2985126R. ISSN: 0022-0302.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20031104
Last Updated on STN: 20031220
Entered Medline: 20031219

L20 ANSWER 5 OF 210 MEDLINE on STN
TI Neutrophil activation and **C-reactive protein** concentration in preeclampsia.
AB Preeclamptic pregnancies seem to be associated with a higher extent of inflammation compared with normal ones. We intended to test this proposal and also to clarify the contribution of some variables in such inflammatory process. We measured total and differential leukocyte count, serum **C-reactive protein** (CRP), and plasma levels of **lactoferrin**, elastase, and granulocyte-macrophage colony-stimulating factor (GM-CSF). Uric acid was also evaluated and used as an indicator of the severity of the disease. A cross-sectional study was performed by evaluating healthy and preeclamptic women in the third trimester of gestation (n = 67 and n = 51, respectively) and 24 to 48 h postpartum (n = 32 and n = 26, respectively). When comparing the third trimester of normal and preeclamptic pregnancies, we found significantly higher levels of uric acid, CRP, and elastase, and a significantly higher elastase to neutrophil ratio in the pathologic group. However, for CRP, statistical significance was lost after adjustment for maternal weight. No significant differences were found in total leukocyte count, plasma levels of GM-CSF, and **lactoferrin** between groups. In preeclampsia, a significant positive correlation was found between

elastase and **lactoferrin** and these neutrophil activation products correlated positively with uric acid level. Considering the analysis of all variables in the postpartum period, only CRP and uric acid levels were significantly elevated in the pathologic group. However, CRP differences obtained in the puerperium seem to be influenced by the increased number of dystocic deliveries in the preeclamptic group. In conclusion, our data suggest that inflammation is further pronounced in preeclampsia and that the extent of neutrophil activation correlates with the severity of this syndrome.

ACCESSION NUMBER: 2003375067 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12908997
TITLE: Neutrophil activation and **C-reactive protein** concentration in preeclampsia.
AUTHOR: Belo Luis; Santos-Silva Alice; Caslake Muriel; Cooney Josephine; Pereira-Leite Luis; Quintanilha Alexandre; Rebelo Irene
CORPORATE SOURCE: Department of Biochemistry, Faculty of Pharmacy, University of Porto, Porto, Portugal.. luis_fbelo@yahoo.com
SOURCE: Hypertension in pregnancy : official journal of the International Society for the Study of Hypertension in Pregnancy, (2003) 22 (2) 129-41.
Journal code: 9421297. ISSN: 1064-1955.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200311
ENTRY DATE: Entered STN: 20030812
Last Updated on STN: 20031218
Entered Medline: 20031126

L20 ANSWER 6 OF 210 MEDLINE on STN
TI Adaptive and inflammatory immune responses in patients infected with strains of Vibrio parahaemolyticus.
AB In patients with diarrhea caused by Vibrio parahaemolyticus, antibody-secreting cell responses to thermostable direct hemolysin (TDH), lipopolysaccharide (LPS), and whole-cell bacteria were seen. TDH- and LPS-specific responses were seen in serum samples, and immunoglobulin A antibody responses were observed in stool. Levels of **C-reactive protein** and nitric oxide metabolites increased in the systemic circulation at the onset of illness. Tumor necrosis factor-alpha and **lactoferrin** levels were high during the acute stage in mucosal secretions and in plasma, whereas interleukin-1beta levels were high only in mucosal secretions. Duodenal and rectal biopsy specimens obtained at the onset of illness showed an acute inflammatory response. The lamina propria showed edema, congestion of blood vessels, and hemorrhage, with an increase in levels of polymorphonuclear neutrophils and macrophages. Strains belonging to different serotypes exhibited varying resistance to killing by serum; the O8:K21 strain was most sensitive. Infection with V. parahaemolyticus results in B cell responses and an acute inflammatory response that is self-limiting.

ACCESSION NUMBER: 2003144949 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12660923
TITLE: Adaptive and inflammatory immune responses in patients infected with strains of Vibrio parahaemolyticus.
AUTHOR: Qadri Firdausi; Alam Muhammad Shamsul; Nishibuchi Mitsuaki; Rahman Taufiqur; Alam Nur Haque; Chisti Jobayer; Kondo Seiichi; Sugiyama Junichi; Bhuiyan Nurul Amin; Mathan Minnie M; Sack David A; Nair G Balakrish
CORPORATE SOURCE: International Centre for Diarrhoeal Disease Research, Bangladesh, GPOBox 128, Dhaka 1000, Bangladesh.. fqadri@icddrb.org
SOURCE: Journal of infectious diseases, (2003 Apr 1) 187 (7) 1085-96. Electronic Publication: 2003-03-19.
Journal code: 0413675. ISSN: 0022-1899.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20030328
Last Updated on STN: 20030501
Entered Medline: 20030430

L20 ANSWER 7 OF 210 MEDLINE on STN
TI Non-invasive investigation of inflammatory bowel disease.
AB The assessment of inflammatory activity in intestinal disease in man can be done using a variety of different techniques. These range from the use of non-invasive acute phase inflammatory markers measured in plasma such as **C reactive protein** (CRP) and the erythrocyte sedimentation rate (ESR) (both of which give an indirect assessment of disease activity) to the direct assessment of disease activity by intestinal biopsy performed during endoscopy in association with endoscopic scoring systems. Both radiology and endoscopy are conventional for the diagnosis of inflammatory bowel disease (IBD). However these techniques have severe limitations when it comes to assessing functional components of the disease such as activity and prognosis. Here we briefly review the value of two emerging intestinal function tests. Intestinal permeability, although ideally suited for diagnostic screening for small bowel Crohn's disease, appears to give reliable predictive data for imminent relapse of small bowel Crohn's disease and it can be used to assess responses to treatment. More significantly it is now clear that single stool assay of neutrophil specific proteins (calprotectin, **lactoferrin**) give the same quantitative data on intestinal inflammation as the 4 day faecal excretion of 111Indium labelled white cells. Faecal calprotectin is shown to be increased in over 95% of patients with IBD and correlates with clinical disease activity. It reliably differentiates between patients with IBD and irritable bowel syndrome. More importantly, at a given faecal calprotectin concentration in patients with quiescent IBD, the test has a specificity and sensitivity in excess of 85% in predicting clinical relapse of disease. This suggests that relapse of IBD is closely related to the degree of intestinal inflammation and suggests that targeted treatment at an asymptomatic stage of the disease may be indicated.
ACCESSION NUMBER: 2002198937 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11819811
TITLE: Non-invasive investigation of inflammatory bowel disease.
AUTHOR: Tibble J A; Bjarnason I
CORPORATE SOURCE: Department of Medicine, Guy's, King's, St Thomas's Medical School, Bessemer Road, London SE5 9PJ, UK.
SOURCE: World journal of gastroenterology : WJG, (2001 Aug) 7 (4) 460-5. Ref: 70
Journal code: 100883448. ISSN: 1007-9327.
PUB. COUNTRY: China
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020405
Last Updated on STN: 20020606
Entered Medline: 20020605

L20 ANSWER 8 OF 210 MEDLINE on STN
TI Increased levels of inflammatory mediators in children and adults infected with Vibrio cholerae O1 and O139.
AB Investigations were carried out to study the production of factors associated with the innate immune response in the systemic and mucosal compartments in adults and children infected with Vibrio cholerae O1 and V. cholerae O139. The levels of nonspecific mediators of the innate defense system, i.e., prostaglandin E(2) (PGE(2)), leukotriene B(4) (LTB(4)), and **lactoferrin** (Lf), as well as myeloperoxidase (MPO), were elevated at the acute stage of the disease in stools obtained from both O1- and O139-infected adults and children. In the systemic compartment, the levels of Lf were increased after onset of disease, which in children remained elevated up to convalescence compared to the healthy

controls. Increased concentrations of **C-reactive protein** were seen in the sera of adult cholera patients at the acute stage of infection. Elevated levels of the nitric oxide (NO*) metabolites (nitrite and nitrate [NO(2)(-) and NO(3)(-)]) were detected in plasma but not in urine. The activity of the scavenger of reactive oxygen species, superoxide dismutase, was higher in the plasma of adults immediately after the onset of disease, suggesting that an active scavenging of reactive oxygen species was taking place. The concentration of 8-iso-prostaglandin F(2 alpha) remained unchanged in the systemic and mucosal compartments in the study subjects. After the recovery of patients from cholera, the concentration of the majority of the metabolites decreased to baseline levels by day 30 after the onset of infection. Immunohistochemical staining showed increased tissue expression of MPO, Lf, and inducible nitric oxide synthase at the acute stage in the duodenal biopsies of adults and rectal biopsies obtained from children with cholera. Very little difference was seen in the levels of the different inflammatory mediators in patients infected with V. cholerae O1 or the encapsulated V. cholerae O139. In summary, these results suggest that elevated concentrations of Lf, MPO, PGE(2), LTB(4), and NO*, as well as other metabolites, during the acute stage of the disease indicate that the innate defense system, as well as the inflammatory process, is activated in both adults and pediatric patients infected with V. cholerae O1 and O139.

ACCESSION NUMBER: 2002138905 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11874856
TITLE: Increased levels of inflammatory mediators in children and adults infected with Vibrio cholerae O1 and O139.
AUTHOR: Qadri Firdausi; Raqib Rubhana; Ahmed Firoz; Rahman Taufiqur; Wenneras Christine; Das Swadesh Kumar; Alam Nur Haque; Mathan Minnie M; Svennerholm Ann-Mari
CORPORATE SOURCE: Centre for Health and Population Research, International Centre for Diarrhoeal Disease Research, Bangladesh, Dhaka 1000, Bangladesh.. fqadri@icddrb.org
SOURCE: Clinical and diagnostic laboratory immunology, (2002 Mar) 9 (2) 221-9.
Journal code: 9421292. ISSN: 1071-412X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020305
Last Updated on STN: 20020612
Entered Medline: 20020611

L20 ANSWER 9 OF 210 MEDLINE on STN
TI Local inflammatory responses following bronchial endotoxin instillation in humans.
AB To study local lung inflammation, 34 subjects had endotoxin (1-4 ng/kg) instilled into a lung segment and saline instilled into a contralateral segment followed by bronchoalveolar lavage (BAL) at 2 h, 6 h, 24 h, or 48 h. Endotoxin instillation resulted in a focal inflammatory response with a distinct time course. An early phase (2 h to 6 h) revealed an increase in neutrophils ($p = 0.0001$) with elevated cytokines (tumor necrosis factor [TNF]-alpha, TNF receptors [TNFR], interleukin [IL]-1beta, IL-1 receptor antagonist, IL-6, granulocyte-colony-stimulating factor [G-CSF], all $p <$ or $= 0.002$, but no change in IL-10) and chemokines (IL-8, epithelial neutrophil activating protein-78, monocyte chemotactic protein-1, macrophage inflammatory protein [MIP]-1alpha, MIP-1beta, all $p <$ or $=$ 0.001, but no change in growth-regulated peptide-alpha). A later phase (24 h to 48 h) showed increased neutrophils, macrophages, monocytes, and lymphocytes (all $p <$ or $= 0.02$), and a return to basal levels of most mediators. Elevated levels of inflammatory markers (TNFR(1), TNFR(2), L-selectin, **lactoferrin**, and myeloperoxidase) persisted in the BAL at 48 h ($p <$ or $= 0.001$). Increased permeability to albumin occurred throughout both phases ($p = 0.001$). Blood **C-reactive protein**, serum amyloid A, IL-6, IL-1ra, G-CSF, but not TNF-alpha increased by 8 h (all $p <$ or $= 0.008$). The local pulmonary inflammatory

response to endotoxin has a unique qualitative and temporal profile of inflammation compared with previous reports of intravenous endotoxin challenges. This model provides a means to investigate factors that initiate, amplify, and resolve local lung inflammation.

ACCESSION NUMBER: 2001333979 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11401879
TITLE: Local inflammatory responses following bronchial endotoxin instillation in humans.
COMMENT: Comment in: Am J Respir Crit Care Med. 2001 Jun;163(7):1516-7. PubMed ID: 11401864
AUTHOR: O'Grady N P; Preas H L; Pugin J; Fiuza C; Tropea M; Reda D; Banks S M; Suffredini A F
CORPORATE SOURCE: Critical Care Medicine Department, Warren G. Magnuson Clinical Center, National Institutes of Health, Bethesda, Maryland, USA.
SOURCE: American journal of respiratory and critical care medicine, (2001 Jun) 163 (7) 1591-8.
Journal code: 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010806
Last Updated on STN: 20010806
Entered Medline: 20010802

L20 ANSWER 10 OF 210 MEDLINE on STN

TI Alpha-1-antichymotrypsin and oxidative stress in the peripheral blood from patients with probable Alzheimer disease: a short-term longitudinal study.

AB To evaluate the stability and reproducibility of selected peripheral oxidative stress markers and their possible relation to cognitive performance, three different blood samples were taken at 7- to 10-day intervals from 11 patients with probable Alzheimer disease (AD) and 11 nondemented controls. Blood samples were also collected once from 6 patients with vascular dementia (VD). Alpha-1-antichymotrypsin (ACT), **C-reactive protein** (CRP), glutathione peroxidase (GSH-Px), superoxide dismutase (SOD), **lactoferrin** (LTF), and total lipid peroxidation (LPO) were then measured. Blood levels of ACT and GSH-Px were increased in AD patients but not in patients with VD. Levels of LTF, CRP, and LPO were comparable between AD patients and controls. Erythrocyte SOD activity was increased in AD patients. Blood levels of ACT negatively correlated with LPO levels and positively correlated with scores of the Global Deterioration Scale of AD patients. ACT might be implicated in controlling oxidative damage of blood lipids and their turnover during the progression of AD.

ACCESSION NUMBER: 2001245993 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11236825
TITLE: Alpha-1-antichymotrypsin and oxidative stress in the peripheral blood from patients with probable Alzheimer disease: a short-term longitudinal study.
AUTHOR: Licastro F; Pedrini S; Davis L J; Caputo L; Tagliabue J; Savorani G; Cucinotta D; Annoni G
CORPORATE SOURCE: Dipartimento di Patologia Sperimentale, University of Bologna, Italy.
SOURCE: Alzheimer disease and associated disorders, (2001 Jan-Mar) 15 (1) 51-5.
Journal code: 8704771. ISSN: 0893-0341.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010510

L20 ANSWER 11 OF 210 MEDLINE on STN

TI Immunoglobulin isotypes of anti-myeloperoxidase and anti-**lactoferrin** antibodies in patients with collagen diseases.

AB To investigate the prevalence and clinical relevance of immunoglobulin (Ig) isotypes of antimyeloperoxidase (MPO) and antilactoferrin (LF) antibodies in collagen diseases, enzyme-linked immunosorbent assay was employed to detect the Ig isotypes of both antibodies. The purified proteins of MPO and LF were used as two major representative antigens for anti-neutrophil cytoplasmic antibodies (ANCA) with a perinuclear staining pattern by an indirect immunofluorescent technique. We examined 131 serum samples from 79 patients with rheumatoid arthritis (RA), 32 with systemic lupus erythematosus (SLE), 14 with progressive systemic sclerosis (PSS), 6 with polymyositis/dermatomyositis (PM/DM), and 5 with idiopathic crescentic glomerulonephritis who served as positive controls and 36 healthy subjects who served as controls. A limited number of patients with RA (4-10%), SLE (6-9%), and PSS (7-14%) but not PM/DM showed positive IgG or IgA anti-MPO antibody (MPO-ANCA) but not IgM MPO-ANCA. However, 10-20% of RA, 40-60% of SLE, 20-36% of PSS but none of the PM/DM patients showed positive IgG, IgA, or IgM anti-LF antibody (LF-ANCA). MPO- and LF-ANCA positivity in RA patients was correlated with markers of disease activity such as the erythrocyte sedimentation rate, **C-reactive protein**, and serum Ig levels. IgG LF-ANCA but not MPO-ANCA positivity in SLE patients also was correlated with the disease activity index but not with clinical features. Neither MPO- nor LF-ANCA positivity in PSS patients was correlated with any clinical features. Overall, both MPO- and LF-ANCA were found mainly in RA, SLE, and PSS patients but not in PM/DM patients. The Ig isotypes of MPO- and LF-ANCA frequently belonged to both IgG and IgA and rarely to the IgM class. Both MPO- and LF-ANCA positivity reflected disease activity in RA and SLE rather than specific organ involvement.

ACCESSION NUMBER: 2001095335 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10939715
TITLE: Immunoglobulin isotypes of anti-myeloperoxidase and anti-**lactoferrin** antibodies in patients with collagen diseases.
AUTHOR: Chikazawa H; Nishiya K; Matsumori A; Hashimoto K
CORPORATE SOURCE: Second Department of Internal Medicine, Kochi Medical School, Nankoku City, Japan.
SOURCE: Journal of clinical immunology, (2000 Jul) 20 (4) 279-86. Journal code: 8102137. ISSN: 0271-9142.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010201

L20 ANSWER 12 OF 210 MEDLINE on STN

TI Prospective evaluation of the frequency and clinical significance of antineutrophil cytoplasmic and anticardiolipin antibodies in community cases of patients with rheumatoid arthritis.

AB OBJECTIVES: To evaluate the frequencies of antineutrophil cytoplasmic (ANCA), anticardiolipin (aCLA) and anti-beta(2)-glycoprotein 1 antibodies (abeta(2)-GP1A) in rheumatoid arthritis (RA) of limited duration in patients recruited primarily from private practitioners (80%), and to attempt to correlate the presence of these antibodies with certain clinical and/or biological criteria. Patients and methods. Patients (n = 102) with RA evolving for <5 yr (mean 2.2 yr) were recruited. A home evaluation collected clinical data [Ritchie articular index, Health Assessment Questionnaire (HAQ) index, extra-articular manifestations] and blood for biological analyses [**C-reactive protein** (CRP), rheumatoid factor, ANCA, aCLA, abeta(2)-GP1A]. ANCA were detected by indirect immunofluorescence on neutrophils and their specificity was determined by enzyme-linked immunosorbent assay (ELISA) and confirmed by immunoblotting; aCLA and abeta(2)-GP1A were detected by ELISA. RESULTS: Patients had mild RA (Ritchie = 11/78 +/- 9.6; HAQ = 0.79/3 +/- 0.7), probably due to the recruitment procedure. ANCA, aCLA

and abeta(2)-GP1A frequencies were 18.5, 7 and 0%, respectively. Titres of ANCA and aCLA were low. A perinuclear ANCA staining pattern was exclusively observed and **lactoferrin** was shown to be the major antigen recognized. No relationship was found between ANCA and aCLA and/or rheumatoid factor, or any clinical manifestations. ANCA were more common in RA of longer duration (cut-off: 4 yr; P = 0.05) and aCLA were correlated with the CRP level (P = 0.05). CONCLUSIONS: In RA of recent onset, ANCA and aCLA were detected at low titres and frequencies, and were not associated with any clinical manifestations. A longitudinal study is needed to determine whether their early appearance is predictive of subsequent disease severity.

ACCESSION NUMBER: 2000386026 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10852977
TITLE: Prospective evaluation of the frequency and clinical significance of antineutrophil cytoplasmic and anticardiolipin antibodies in community cases of patients with rheumatoid arthritis.
AUTHOR: Vittecoq O; Jouen-Beades F; Krzanowska K; Bichon-Tauvel I; Menard J F; Daragon A; Gilbert D; Tron F; Le Loet X
CORPORATE SOURCE: Service de Rhumatologie, INSERM U 519 et Institut Federatif de Recherche Multidisciplinaire sur les Peptides, Centre Hospitalier Universitaire de Rouen, France.
SOURCE: Rheumatology (Oxford, England), (2000 May) 39 (5) 481-9. Journal code: 100883501. ISSN: 1462-0324.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000818
Last Updated on STN: 20030304
Entered Medline: 20000808

L20 ANSWER 13 OF 210 MEDLINE on STN
TI The inflammatory response following treatment of abdominal aortic aneurysms: a comparison between open surgery and endovascular repair.
AB OBJECTIVES: to compare the inflammatory response following endovascular and conventional AAA repair. Design: prospective study. PATIENTS AND METHODS: ten patients were selected for open surgery (OPEN) and ten for endovascular (ENDO) AAA repair. Leukocytes, platelets, myeloperoxidase, **lactoferrin**, beta-thromboglobulin, **C-reactive protein** (CRP), interleukin 6 (IL-6), tumour necrosis factor alpha (TNF-alpha) and complement activation products were measured before, during and after surgery. RESULTS: in the OPEN group the median hospital stay was longer (6 vs. 12 days, p=0.001) and more patients required transfusion (p=0.02). IL-6 and CRP increased postoperatively, most in OPEN (p<0.01). Platelet counts decreased after the first angiography in ENDO (p<0.01) and before aortic cross-clamping in OPEN (p<0.05). The decrease was larger in OPEN (p=0.02). Leukocyte counts decreased after the first angiography in ENDO, and thereafter increased (p=0.001). An equivalent increase was observed in OPEN after declamping (p=0.001). Leukocyte and platelet degranulation products increased after the first angiography in ENDO and after declamping in OPEN. Changes in complement activation products were small. TNF-alpha did not change significantly. CONCLUSION: endovascular AAA repair caused significant leukocyte and platelet activation. Based on the timing of activation this could be caused by radiographic contrast media.

ACCESSION NUMBER: 2000295332 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10828237
TITLE: The inflammatory response following treatment of abdominal aortic aneurysms: a comparison between open surgery and endovascular repair.
AUTHOR: Odegard A; Lundbom J; Myhre H O; Hatlinghus S; Bergh K; Waage A; Bjerve K S; Mollnes T E; Aadahl P; Lie T A; Videm V
CORPORATE SOURCE: Department of Radiology, Regional Hospital of Trondheim, Norway.

SOURCE: European journal of vascular and endovascular surgery : official journal of the European Society for Vascular Surgery, (2000 May) 19 (5) 536-44.
Journal code: 9512728. ISSN: 1078-5884.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000720
Last Updated on STN: 20000720
Entered Medline: 20000710

L20 ANSWER 14 OF 210 MEDLINE on STN
TI Heparin-coated cardiopulmonary bypass circuits reduce circulating complement factors and interleukin-6 in paediatric heart surgery.
AB Children are sensitive to the inflammatory side effects of cardiopulmonary bypass (CPB). Our intention was to investigate if the biocompatibility benefits of heparin-coated CPB circuits apply to children. In 20 operations, 19 children were randomized to heparin-coated (group HC, n = 10) or standard (group C, n = 10) bypass circuits. Plasma levels of acute phase reactants, interleukins, granulocytic proteins and complement factors were measured. All were significantly elevated after CPB. Levels of complement factor C3a (851 (791-959)ng/ml [median with quartiles] in group C, 497 (476-573)ng/ml in group HC, $p < 0.001$), Terminal Complement Complex (114 (71-130) AU/ml in group C, 35.5 (28.9-51.4) AU/ml in group HC, $p < 0.001$), and interleukin-6 (570 (203-743) pg/ml in group C, 168 (111-206)pg/ml in group HC, $p = 0.005$), were significantly reduced in group HC. Heparin-coated CPB circuits improve the biocompatibility of CPB during heart surgery in the paediatric patient population, as reflected by significantly reduced levels of circulating complement factors and interleukin-6.
ACCESSION NUMBER: 2000273542 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10816058
TITLE: Heparin-coated cardiopulmonary bypass circuits reduce circulating complement factors and interleukin-6 in paediatric heart surgery.
AUTHOR: Olsson C; Siegbahn A; Henze A; Nilsson B; Venge P; Joachimsson P O; Thelin S
CORPORATE SOURCE: Department of Cardiothoracic Surgery, Uppsala University Hospital, Sweden.
SOURCE: Scandinavian cardiovascular journal : SCJ, (2000) 34 (1) 33-40.
Journal code: 9708377. ISSN: 1401-7431.
PUB. COUNTRY: Norway
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000728
Last Updated on STN: 20000728
Entered Medline: 20000714

L20 ANSWER 15 OF 210 MEDLINE on STN
TI Impaired neutrophil exocytosis in patients with severe pneumonia.
AB OBJECTIVE: Polymorphonuclear neutrophils (PMN) are one of the major effector cells of pulmonary defence against bacterial infection. To determine whether neutrophil function is impaired in patients with severe pneumonia, we assessed the two main partial functions exocytosis and oxidative response (ROS production) in isolated neutrophils from the peripheral venous blood of pneumonia patients and healthy volunteers. In addition, pulmonary neutrophils and peripheral neutrophils were compared in pneumonia patients. PATIENTS AND METHODS: Twenty-one patients with severe pneumonia were enrolled in the study. Eleven patients were mechanically ventilated, ten patients breathed spontaneously. For comparison, ten healthy adults were studied. The release of two markers

of neutrophil exocytosis, **lactoferrin** and myeloperoxidase (MPO), with and without stimulation by phorbol-myristate-acetate (PMA), was determined using immunoluminometric assays. ROS production was quantified using luminol-enhanced chemiluminescence. In addition, the clinical severity of pneumonia was correlated to neutrophil exocytosis. RESULTS: With regard to blood neutrophils, both basal and PMA-stimulated exocytosis were significantly impaired in pneumonia patients compared to healthy volunteers (basal **lactoferrin** secretion in pneumonia patients: 0.25+/-0.36 pg/PMN versus controls: 1.17+/-0.78 pg/PMN, p<0.01). In contrast, both basal and PMA-stimulated ROS production were increased in patients compared to controls (spontaneous chemiluminescence in pneumonia patients: 13.6x10(5) cpm versus controls: 5.5x10(5) cpm). In pneumonia patients, the pulmonary neutrophils released significantly more **lactoferrin**, MPO and ROS compared to blood neutrophils (basal **lactoferrin** secretion of pulmonary neutrophils: 1.19+/-1.55 pg/PMN; p<0,01). However, after stimulation with PMA the exocytosis of pulmonary and blood neutrophils was similar. The severity of pneumonia and prognostic indices like albumin were inversely correlated to the release of **lactoferrin** in blood neutrophils (p<0,05). CONCLUSION: In patients with severe pneumonia, the exocytosis of blood neutrophils was significantly impaired. In contrast to this, the oxidative response was increased. Impaired bone marrow maturation of neutrophils during severe infection, perhaps due to shortened maturation time, could explain these findings.

ACCESSION NUMBER: 1999158364 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10051077
TITLE: Impaired neutrophil exocytosis in patients with severe pneumonia.
AUTHOR: Zimmermann B; Dalhoff K; Braun J
CORPORATE SOURCE: Department of Medicine II, Medical University of Lubeck, Germany.
SOURCE: Intensive care medicine, (1999 Jan) 25 (1) 44-51. Journal code: 7704851. ISSN: 0342-4642.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990601
Last Updated on STN: 19990601
Entered Medline: 19990520

=> d his

(FILE 'HOME' ENTERED AT 15:53:22 ON 16 MAR 2005)

FILE 'MEDLINE, JAPIO, BIOSIS, WPIDS, JICST-EPLUS, BIOTECHDS, HCAPLUS, SCISEARCH, CEN, CEABA-VTB, BIOBUSINESS, EMBASE, DGENE' ENTERED AT 15:54:03 ON 16 MAR 2005

L1 1 S LACTOFERRIN AND (REDUCE CIRCULATING LEVELS OF CHOLESTEROL?)
L2 0 S LACTOFERRIN COMPOSITION ADJ2 ADMINISTRATION
L3 18 S LACTOFERRIN COMPOSITION
L4 26082 S LACTOFERRIN
L5 0 S L4 AND (REDUCE VASCULAR INFLAMMATION?)
L6 25 S HEART DISEASE AND L4
L7 205 S L4 AND DIABETES
L8 51 S L4 AND HYPERTENSION
L9 38 S L7 AND TREATMENT
L10 16 S L8 AND TREATMENT
L11 0 S LACTOFERRIN ADJ2 CHOLESTEROL
L12 1555 S L4 AND THERAPY
L13 3013 S L4 AND TREATMENT
L14 267 S L4 AND HEART
L15 33 S L14 AND L13
L16 8 S L15 AND L12
E VARADHACHARY, A/AU
E ENGELMAYER, J/AU

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                E GLYNN,P/AU
                E WANG, Y/AU
L17             0 S LDL DECREASE AND L4
L18             0 S DECREASE TRIGLYCERIDE AND L4
L19             0 S DECREASE VLDL AND L4
L20           210 S (C-REACTIVE PROTEIN) AND L4

=> s l4 and (treatment or theraphy or medicament or injection)
L21        3581 L4 AND (TREATMENT OR THERAPHY OR MEDICAMENT OR INJECTION)

=> s l21 and heart
L22          51 L21 AND HEART

=> s l2 and atherosclerosis
L23           0 L2 AND ATHEROSCLEROSIS

=> d l22 ti abs ibib 1-10

L22  ANSWER 1 OF 51     MEDLINE on STN
TI   Bovine lactoferrin has a nitric oxide-dependent hypotensive
     effect in rats.
AB   Lactoferrin (LF) is a multifunctional protein that is found in
     milk, neutrophils, and other biological fluids. Under inflammatory
     conditions, LF production is increased in the periphery by neutrophils.
     However, the cardiovascular function of LF is still unknown. In the
     present study, we investigated the effect of bovine LF (BLF) on the mean
     blood pressure (MBP) and heart rate (HR) in urethane-
     anesthetized rats and the vascular function of BLF in the rat thoracic
     aorta. Intravenous injection of BLF produced dose-dependent
     decreases in MBP but did not affect HR, while the opioid agonist morphine
     decreased both MBP and HR. The hypotensive effect of BLF was not altered
     by naloxone methiodide, which cannot pass through the blood-brain barrier,
     but was significantly reduced by naloxone hydrochloride, which does pass
     through the blood-brain barrier. BLF-induced hypotension was completely
     blocked by the NO synthase inhibitor NG-nitro-l-arginine methyl ester
     (l-NAME) but not by the inactive enantiomer of l-NAME, NG-nitro-d-arginine
     methyl ester (d-NAME). BLF-induced hypotension was not altered by the
     muscarinic ACh receptor antagonist atropine or the cyclooxygenase
     inhibitor diclofenac. BLF produced relaxation in endothelium-intact but
     not endothelium-denuded aortic rings precontracted with phenylephrine.
     The relaxation evoked by BLF was completely blocked by l-NAME but not by
     d-NAME or the ATP-sensitive potassium channel blocker glibenclamide.
     These results suggest that BLF causes hypotension via an
     endothelium-dependent vasodilation that is strongly mediated by NO
     production and that BLF-induced hypotension also may be mediated by the
     central opioidergic system.
ACCESSION NUMBER:    2004009027     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 14563657
TITLE:               Bovine lactoferrin has a nitric oxide-dependent
                     hypotensive effect in rats.
AUTHOR:              Hayashida Ken-Ichiro; Takeuchi Takashi; Ozaki Takeshi;
                     Shimizu Hirohiko; Ando Kunio; Miyamoto Atsushi; Harada
                     Etsumori
CORPORATE SOURCE:    Dept. of Veterinary Physiology, Faculty of Agriculture,
                     Tottori Univ., Tottori 680-0945, Japan.
SOURCE:              American journal of physiology. Regulatory, integrative and
                     comparative physiology, (2004 Feb) 286 (2) R359-65.
                     Electronic Publication: 2003-10-16.
                     Journal code: 100901230. ISSN: 0363-6119.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200402
ENTRY DATE:          Entered STN: 20040107
                     Last Updated on STN: 20040302
                     Entered Medline: 20040227
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L22 ANSWER 2 OF 51 MEDLINE on STN
TI Molecular cloning and functional expression of a human intestinal **lactoferrin** receptor.
AB **Lactoferrin** (Lf), a major iron-binding protein in human milk, has been suggested to have multiple biological roles such as facilitating iron absorption, modulating the immune system, embryonic development, and cell proliferation. Our previous binding studies suggested the presence of a specific receptor for Lf (LfR) in the small intestine of newborn infants, which may facilitate iron absorption. We here report the cloning and the functional expression of the human intestinal LfR and the evidence of its involvement in iron metabolism. The entire coding region of the LfR cDNA was cloned by PCR based on amino acid sequences of the purified native LfR (nLfR). The recombinant LfR (rLfR) was then expressed in a baculovirus-insect cell system and purified by immobilized human Lf (hLf) affinity chromatography where binding of hLf to the rLfR was partially Ca(2+) dependent. The apparent molecular mass was 136 kDa under nonreducing conditions and 34 kDa under reducing conditions. 125I-hLf bound to the rLfR with an apparent K(d) of approximately 360 nM. These biochemical properties of the rLfR are similar to those of the nLfR. RT-PCR revealed that the gene was expressed at high levels in fetal small intestine and in adult **heart** and at lower levels in Caco-2 cells. PI-PLC **treatment** of Caco-2 cells indicated that the LfR is GPI anchored. In Caco-2 cells transfected with the LfR gene, 125I-hLf binding and 59Fe-hLf uptake were increased by 1.7 and 3.4 times, respectively, compared to those in mock-transfected cells. Our findings demonstrate the presence of a unique receptor-mediated mechanism for nutrient uptake by the newborn.
ACCESSION NUMBER: 2001699646 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11747454
TITLE: Molecular cloning and functional expression of a human intestinal **lactoferrin** receptor.
AUTHOR: Suzuki Y A; Shin K; Lonnerdal B
CORPORATE SOURCE: Department of Nutrition, University of California, Davis, One Shields Avenue, Davis, California 95616, USA.
SOURCE: Biochemistry, (2001 Dec 25) 40 (51) 15771-9. Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF271386
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011219
Last Updated on STN: 20020128
Entered Medline: 20020123

L22 ANSWER 3 OF 51 MEDLINE on STN
TI **Lactoferrin** and anti-**lactoferrin** antibodies: effects of ironloading of **lactoferrin** on albumin extravasation in different tissues in rats.
AB **Lactoferrin** is a cationic iron-binding protein, which is released from activated neutrophils in concert with reactive oxygen species. In vitro, **lactoferrin** has both anti- and proinflammatory effects; many of them dependent on iron-binding. In vivo, only iron-free **lactoferrin** reduced inflammatory hyperpermeability in the lung. We therefore examined whether 1 mg iron-free (Apo-Lf) or iron-saturated **lactoferrin** (Holo-Lf) alone or followed by anti-**lactoferrin** antibodies (aLf) affected permeability evaluated by extravasation of radiolabelled bovine serum albumin (CBSA) in different tissues of anaesthetized rats. Fifteen minutes after i.v. **injection** of Lf, aLf or saline was given and circulatory arrest was induced 20 min thereafter. Measurements were performed in control, after Apo-Lf, Holo-Lf, Apo-Lf + aLf, Holo-Lf + aLf and aLf alone (n=6-8 in each group). No intergroup differences were found for plasma volume and haematocrit at the start and end of the 37 min extravasation period or for total tissue water in any of the six different tissues studied, excluding larger transcapillary fluid shifts. However, increases in CBSA were seen without differences in tissue intravascular

volume. Iron-free **lactoferrin** and aLf alone did not change CBSA significantly. Iron-saturated **lactoferrin** significantly increased CBSA in skin (neck), trachea and left ventricle of the **heart** to 249 +/- 9, 284 +/- 16 and 160 +/- 7% of control, respectively. When followed by aLf, both Apo- and Holo-Lf increased CBSA significantly in four and five of the tissues studied, respectively. However, no significant effect was seen for Holo-Lf + aLf compared with Holo-Lf alone. In conclusion, iron-saturated, but not iron-free **lactoferrin** increased CBSA, whereas antilactoferrin increased CBSA compared with **lactoferrin** alone only when following iron-free **lactoferrin**.

ACCESSION NUMBER: 2001031974 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10971218
TITLE: **Lactoferrin** and anti-**lactoferrin** antibodies: effects of ironloading of **lactoferrin** on albumin extravasation in different tissues in rats.
AUTHOR: Erga K S; Peen E; Tenstad O; Reed R K
CORPORATE SOURCE: Department of Physiology, University of Bergen, Norway.
SOURCE: Acta physiologica Scandinavica, (2000 Sep) 170 (1) 11-9.
Journal code: 0370362. ISSN: 0001-6772.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001120

L22 ANSWER 4 OF 51 MEDLINE on STN
TI Heparin-coated cardiopulmonary bypass circuits reduce circulating complement factors and interleukin-6 in paediatric **heart** surgery.
AB Children are sensitive to the inflammatory side effects of cardiopulmonary bypass (CPB). Our intention was to investigate if the biocompatibility benefits of heparin-coated CPB circuits apply to children. In 20 operations, 19 children were randomized to heparin-coated (group HC, n = 10) or standard (group C, n = 10) bypass circuits. Plasma levels of acute phase reactants, interleukins, granulocytic proteins and complement factors were measured. All were significantly elevated after CPB. Levels of complement factor C3a (851 (791-959)ng/ml [median with quartiles] in group C, 497 (476-573)ng/ml in group HC, $p < 0.001$), Terminal Complement Complex (114 (71-130) AU/ml in group C, 35.5 (28.9-51.4) AU/ml in group HC, $p < 0.001$), and interleukin-6 (570 (203-743) pg/ml in group C, 168 (111-206)pg/ml in group HC, $p = 0.005$), were significantly reduced in group HC. Heparin-coated CPB circuits improve the biocompatibility of CPB during **heart** surgery in the paediatric patient population, as reflected by significantly reduced levels of circulating complement factors and interleukin-6.

ACCESSION NUMBER: 2000273542 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10816058
TITLE: Heparin-coated cardiopulmonary bypass circuits reduce circulating complement factors and interleukin-6 in paediatric **heart** surgery.
AUTHOR: Olsson C; Siegbahn A; Henze A; Nilsson B; Venge P; Joachimsson P O; Thelin S
CORPORATE SOURCE: Department of Cardiothoracic Surgery, Uppsala University Hospital, Sweden.
SOURCE: Scandinavian cardiovascular journal : SCJ, (2000) 34 (1) 33-40.
Journal code: 9708377. ISSN: 1401-7431.
PUB. COUNTRY: Norway
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007

ENTRY DATE: Entered STN: 20000728
Last Updated on STN: 20000728
Entered Medline: 20000714

L22 ANSWER 5 OF 51 MEDLINE on STN
TI Characterization of the binding of ferritin to the rat liver ferritin receptor.
AB The binding characteristics and specificity of the rat hepatic ferritin receptor were investigated using ferritins prepared from rat liver, **heart**, spleen, kidney and serum, human liver and serum, guinea pig liver and horse spleen as well as ferritins enriched with respect to either H- or L-type subunit composition, prepared by chromatofocusing of rat liver ferritin on Mono-P or by reverse-phase chromatography of ferritin subunits on ProRPC 5/10. No significant difference was apparent in the binding of any of the tissue ferritins, or of ferritins of predominantly acidic or basic subunit composition. However, serum ferritin bound with a lower affinity. The effect of carbohydrate on the ferritin-receptor binding was examined by glycosidase **treatment** of tissue and serum ferritins. Tissue ferritin binding was unaffected, while serum ferritin binding affinity was increased to that of the tissue ferritins. Inhibition of ferritin binding by **lactoferrin** was not due to common carbohydrate moieties as previously suggested but was due to direct binding of **lactoferrin** to ferritin. Therefore, carbohydrate residues do not appear to facilitate receptor-ferritin binding, and sialic acid residues present on serum ferritin may in fact interfere with binding. The results indicate that the hepatic ferritin receptor acts preferentially to remove tissue ferritins from the circulation. The lower binding affinity of serum ferritin for the ferritin receptor explains its slower in vivo clearance relative to tissue ferritins.
ACCESSION NUMBER: 86051611 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2998476
TITLE: Characterization of the binding of ferritin to the rat liver ferritin receptor.
AUTHOR: Mack U; Storey E L; Powell L W; Halliday J W
SOURCE: Biochimica et biophysica acta, (1985 Dec 13) 843 (3) 164-70.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198601
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19860121

L22 ANSWER 6 OF 51 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Bovine **lactoferrin** has a nitric oxide-dependent hypotensive effect in rats.
AB **Lactoferrin** (LF) is a multifunctional protein that is found in milk, neutrophils, and other biological fluids. Under inflammatory conditions, LF production is increased in the periphery by neutrophils. However, the cardiovascular function of LF is still unknown. In the present study, we investigated the effect of bovine LF (BLF) on the mean blood pressure (MBP) and **heart** rate (HR) in urethane-anesthetized rats and the vascular function of BLF in the rat thoracic aorta. Intravenous **injection** of BLF produced dose-dependent decreases in MBP but did not affect HR, while the opioid agonist morphine decreased both MBP and HR. The hypotensive effect of BLF was not altered by naloxone methiodide, which cannot pass through the blood-brain barrier, but was significantly reduced by naloxone hydrochloride, which does pass through the blood-brain barrier. BLF-induced hypotension was completely blocked by the NO synthase inhibitor NG-nitro-L-arginine methyl ester (L-NAME) but not by the inactive enantiomer of L-NAME, NG-nitro-D-arginine methyl ester (D-NAME). BLF-induced hypotension was not altered by the muscarinic ACh receptor antagonist atropine or the cyclooxygenase inhibitor diclofenac. BLF produced relaxation in endothelium-intact but

not endothelium-denuded aortic rings precontracted with phenylephrine. The relaxation evoked by BLF was completely blocked by L-NAME but not by D-NAME or the ATP-sensitive potassium channel blocker glibenclamide. These results suggest that BLF causes hypotension via an endothelium-dependent vasodilation that is strongly mediated by NO production and that BLF-induced hypotension also may be mediated by the central opioidergic system.

ACCESSION NUMBER: 2004:143975 BIOSIS
DOCUMENT NUMBER: PREV200400144150
TITLE: Bovine **lactoferrin** has a nitric oxide-dependent hypotensive effect in rats.
AUTHOR(S): Hayashida, Ken-ichiro; Takeuchi, Takashi; Ozaki, Takeshi; Shimizu, Hirohiko; Ando, Kunio; Miyamoto, Atsushi; Harada, Etsumori [Reprint Author]
CORPORATE SOURCE: Dept. of Veterinary Physiology, Faculty of Agriculture, Tottori Univ., Tottori, 680-0945, Japan harada@muses.tottori-u.ac.jp
SOURCE: American Journal of Physiology, (February 2004) Vol. 286, No. 2 Part 2, pp. R359-R365. print. ISSN: 0002-9513 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 2004 Last Updated on STN: 10 Mar 2004

L22 ANSWER 7 OF 51 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Molecular cloning and functional expression of a human intestinal **lactoferrin** receptor.
AB **Lactoferrin** (Lf), a major iron-binding protein in human milk, has been suggested to have multiple biological roles such as facilitating iron absorption, modulating the immune system, embryonic development, and cell proliferation. Our previous binding studies suggested the presence of a specific receptor for Lf (LfR) in the small intestine of newborn infants, which may facilitate iron absorption. We here report the cloning and the functional expression of the human intestinal LfR and the evidence of its involvement in iron metabolism. The entire coding region of the UR cDNA was cloned by PCR based on amino acid sequences of the purified native UR (nLfR). The recombinant UR (rLfR) was then expressed in a baculovirus-insect cell system and purified by immobilized human Lf (hLf) affinity chromatography where binding of hLf to the rLfR was partially Ca2+ dependent. The apparent molecular mass was 136 kDa under nonreducing conditions and 34 kDa under reducing conditions. 125I-hLf bound to the rLfR with an apparent Kd of apprx360 nM. These biochemical properties of the rLfR are similar to those of the nLfR. RT-PCR revealed that the gene was expressed at high levels in fetal small intestine and in adult **heart** and at lower levels in Caco-2 cells. PI-PLC **treatment** of Caco-2 cells indicated that the UR is GPI anchored. In Caco-2 cells transfected with the LfR gene, 125I-hLf binding and 59Fe-hLf uptake were increased by 1.7 and 3.4 times, respectively, compared to those in mock-transfected cells. Our findings demonstrate the presence of a unique receptor-mediated mechanism for nutrient uptake by the newborn.

ACCESSION NUMBER: 2002:100410 BIOSIS
DOCUMENT NUMBER: PREV200200100410
TITLE: Molecular cloning and functional expression of a human intestinal **lactoferrin** receptor.
AUTHOR(S): Suzuki, Yasushi A.; Shin, Kouichirou; Lonnerdal, Bo [Reprint author]
CORPORATE SOURCE: Department of Nutrition, University of California, Davis, One Shields Avenue, Davis, CA, 95616, USA bllonnerdal@ucdavis.edu
SOURCE: Biochemistry, (December 25, 2001) Vol. 40, No. 51, pp. 15771-15779. print. CODEN: BICHAW. ISSN: 0006-2960.
DOCUMENT TYPE: Article
LANGUAGE: English
OTHER SOURCE: Genbank-AW029086; Genbank-NM010584; Genbank-R06009
ENTRY DATE: Entered STN: 24 Jan 2002

Last Updated on STN: 21 Mar 2002

L22 ANSWER 8 OF 51 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI **Lactoferrin** and anti-**lactoferrin** antibodies: Effects of ironloading of **lactoferrin** on albumin extravasation in different tissues in rats.
AB **Lactoferrin** is a cationic iron-binding protein, which is released from activated neutrophils in concert with reactive oxygen species. In vitro, **lactoferrin** has both anti- and proinflammatory effects; many of them dependent on iron-binding. In vivo, only iron-free **lactoferrin** reduced inflammatory hyperpermeability in the lung. We therefore examined whether 1 mg iron-free (Apo-Lf) or iron-saturated **lactoferrin** (Holo-Lf) alone or followed by anti-**lactoferrin** antibodies (aLf) affected permeability evaluated by extravasation of radiolabelled bovine serum albumin (CBSA) in different tissues of anaesthetized rats. Fifteen minutes after i.v. **injection** of Lf, aLf or saline was given and circulatory arrest was induced 20 min thereafter. Measurements were performed in control, after Apo-Lf, Holo-Lf, Apo-Lf + aLf, Holo-Lf + aLf and aLf alone (n = 6-8 in each group). No intergroup differences were found for plasma volume and haematocrit at the start and end of the 37 min extravasation period or for total tissue water in any of the six different tissues studied, excluding larger transcapillary fluid shifts. However, increases in CBSA were seen without differences in tissue intravascular volume. Iron-free **lactoferrin** and aLf alone did not change CBSA significantly. Iron-saturated **lactoferrin** significantly increased CBSA in skin (neck), trachea and left ventricle of the **heart** to 249 +- 9, 284 +- 16 and 160 +- 7% of control, respectively. When followed by aLf, both Apo- and Holo-Lf increased CBSA significantly in four and five of the tissues studied, respectively. However, no significant effect was seen for Holo-Lf + aLf compared with Holo-Lf alone. In conclusion, iron-saturated, but not iron-free **lactoferrin** increased CBSA, whereas antilactoferrin increased CBSA compared with **lactoferrin** alone only when following iron-free **lactoferrin**.
ACCESSION NUMBER: 2000:521892 BIOSIS
DOCUMENT NUMBER: PREV200000521892
TITLE: **Lactoferrin** and anti-**lactoferrin** antibodies: Effects of ironloading of **lactoferrin** on albumin extravasation in different tissues in rats.
AUTHOR(S): Erga, K. S. [Reprint author]; Peen, E.; Tenstad, O.; Reed, R. K.
CORPORATE SOURCE: Department of Physiology, University of Bergen, Arstadveien 19, N-5009, Bergen, Norway
SOURCE: Acta Physiologica Scandinavica, (September, 2000) Vol. 170, No. 1, pp. 11-19. print.
CODEN: APSCAX. ISSN: 0001-6772.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Nov 2000
Last Updated on STN: 11 Jan 2002

L22 ANSWER 9 OF 51 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI General pharmacological properties of partially degraded **lactoferrin**-derived peptide (MONL-03).
AB The general pharmacological properties of MONL-03, a fragment of **lactoferrin** which is a whey protein in milk, were studied in mice and rats. In mice, intracerebroventricular **injection** of MONL-03 (0.5-20 mu-g/mouse) increased ambulation and the electrical conductance of the paw pads, but it did not alter sleeping time after pentobarbital. Single i.p. **injection** of MONL-03 (10-50 mg/kg) decreased ambulation and the electrical conductance, and it prolonged sleeping time after pentobarbital. Oral administration of MONL-03 (100, 200 mg/kg), however, did not markedly affect ambulation. Single i.p. **injection** of MONL-03 (10-50 mg/kg) prolonged the duration of gasping response and decreased the mortality caused by coadministration of adrenaline and collagen. Intradermal **injection** of MONL-03 (1 mg/mouse) induced edema. In anesthetized mice, **heart** rate

decreased after i.p. **injection** of MONL-03 (10-50 mg/kg). In anesthetized rats, i.v. **injection** of MONL-03 (0.5, 1 mg/kg) antagonized depression of the S wave in the electrocardiogram evoked by vasopressin. Single i.v. **injection** of MONL-03 caused a transient fall in the systolic blood pressure. This effect of MONL-03 was diminished by previous administration of heparin. These results suggest that MONL-03 has various pharmacological actions which are relatively weak on oral administration.

ACCESSION NUMBER: 1996:322370 BIOSIS
DOCUMENT NUMBER: PREV199699044726
TITLE: General pharmacological properties of partially degraded **lactoferrin**-derived peptide (MONL-03).
AUTHOR(S): Orikasa, Shuzo; Shimamura, Seiichi
CORPORATE SOURCE: Biochem. Res. Lab., Morinaga Milk Ind. Co. Ltd., 1-83 Higashihara 5-chome, Zama, Kanagawa 228, Japan
SOURCE: Oyo Yakuri, (1996) Vol. 51, No. 5, pp. 253-261.
CODEN: OYYAA2. ISSN: 0300-8533.
DOCUMENT TYPE: Article
LANGUAGE: Japanese
ENTRY DATE: Entered STN: 11 Jul 1996
Last Updated on STN: 11 Jul 1996

L22 ANSWER 10 OF 51 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI CHARACTERIZATION OF THE BINDING OF FERRITIN TO THE RAT LIVER FERRITIN RECEPTOR.
AB The binding characteristics and specificity of the rat hepatic ferritin receptor was investigated using ferritins prepared from rat liver, **heart**, spleen, kidney and serum, human liver and serum, guinea pig liver and horse spleen as well as ferritins enriched with respect to either H- or L-type subunit composition, prepared by chromatofocusing of rat liver ferritin on Mono-P or by reverse-phase chromatography of ferritin subunits on ProRPC 5/10. No significant difference was apparent in the binding of any of the tissue ferritins, or of ferritins of predominantly acidic or basic subunit composition. However, serum ferritin bound with a lower afinity. The effect of carbohydrate on the ferritin-receptor binding was examined by glycosidase **treatment** of tissue and serum ferritins. Tissue ferritin binding was unaffected, while serum ferritin binding affinity was increased to that of the tissue ferritins. Inhibition of ferritin binding by **lactoferrin** was not due to common carbohydrate moieties as previously suggested but was due to direct binding of **lactoferrin** to ferritin. Therefore, carbohydrate residues do not appear to facilitate receptor-ferritin binding, and sialic acid residues present on serum ferritin may in fact interfere with binding. The results indicate that the hepatic ferritin receptor acts preferentially to remove tissue ferritins from the circulation. The lower binding affinity of serum ferritin for the ferritin receptor explains its slower in vivo clearance relative to tissue ferritins.

ACCESSION NUMBER: 1986:169936 BIOSIS
DOCUMENT NUMBER: PREV198681080352; BA81:80352
TITLE: CHARACTERIZATION OF THE BINDING OF FERRITIN TO THE RAT LIVER FERRITIN RECEPTOR.
AUTHOR(S): MACK U [Reprint author]; STOREY E L; POWELL L W; HALLIDAY J W
CORPORATE SOURCE: DEP MED, CLINICAL SCI BUILDING, ROYAL BRISBANE HOSP, BRISBANE, QLD 4029, AUST
SOURCE: Biochimica et Biophysica Acta, (1985) Vol. 843, No. 3, pp. 164-170.
CODEN: BBACAQ. ISSN: 0006-3002.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 26 Apr 1986
Last Updated on STN: 26 Apr 1986